A

# CLINICAL AND EXPERIMENTAL STUDY

of

# INFANTILE DIARRHOEA

from a

Dietetic and Therapeutic point of view.

THESIS

for the Degree of

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Vol. I.

# INTRODUCTION.

## Introduction.

In spite of the great advances which, of late years, have been achieved in the prevention and cure of disease the mortality among infants has remained undiminished. Every year, diarrhoea, the most fatal scourge of infancy, has been allowed to exact its appalling toll of human lives and medical science has hitherto stood powerless before this annual holocaust But, a new era has now dawned; the genius of the late Professor Budin has completely revolutionised the subject of the infantile mortality in France and his methods are rapidly spreading throughout the civilised world. As M'Cosh scholar I was privileged to be one of his students and to his inspiration and aid is due much of this humble endeavour to contribute to our greater knowledge of infantile diarrhoea, its cause, treatment and prevention.

The scope of the Thesis is outlined in the table of contents but I would, here, like briefly to refer to several parts to which I have devoted special attention.

First, I wish to lay particular stress on the great value of infusion as a measure which tides over critical /

critical periods and allows time for the exhibition of more permanent remedies. Formerly, there was no proper infusing apparatus; the saline was not sufficiently protected from contamination; it could not be maintained at the desired temperature during the fifteen to forty five minutes which the procedure occupies; and the amount given could not be accurately determined. I have described a simple infusing apparatus which I devised (pages 70-73) to render the operation simpler, safer, shorter and more efficacious. It is depicted on plate V which faces page 71.

I wish to emphasise the great value of saline infusion when reinforced by the addition of adrenalin chloride.

Where this treatment was employed I have indicated in red on the charts which also show the curve of the opsonic index (green) ; and it is note worthy how often infusion was immediately followed by a rise in the opsonic index.

Among curative measures I have given special prominence to an experimental therapeutic and dietetic method, which I had the opportunity of exhaustively /

exhaustively studying.

Hitherto, the importance of diminishing the toxic absorption from the alimentary canal has led us, to exalt drugs which exert an antiseptic action on the bowel and its contents to the first position among therapeutic measures. Faith in them has remained unshaken in spite of results. In acute cases time is of vital importance and no chemical antiseptic can quickly and satisfactorily modify intestinal fermentation, in doses which will not induce general toxic and local irritant effects. We, therefore, tried a bacterial antiseptic which was more pathogenic to man. We administered to twenty one cases a stated dose of a milk culture of lactic acid bacilli. These bacilli, in addition to their powerful effect upon the intestinal flora, so changed the chemical composition of the milk in which they had grown that infants were able to retain and assimilate it. The intolerance to food of infants suffering from acute diarrhoea has hitherto dictated a dietetic treatment, which consisted mainly in bold starvation but in this remedy to which I gave the name of "Lactated Milk" - we are /

are enabled to administer a fluid of considerable nutritive value during the very height of the disease.

I have impartially detailed and discussed my successes and failures among the twenty one cases comprised in this experiment (pages 86-119). The results encourage the hope that with greater experience of this remedy we may yet be able successfully to treat the great majority of cases of acute infantile diarrhoea.

I gave an exhaustive trial to normal Horse Serum. In it, I thought, I had a remedy which, injected subcutaneously, would not only have a considerable nutritive value - and thus be of especial service in this disease where assimilation is practically arrested - but also a decided effect in raising the resistance of the patient. Theoretically it is an ideal method of treatment for gaining time, but its results were most disappointing. The opsonic index to B. Coli Communis was taken to afford a test of its influence. Two typical cases were first investigated without Horse Serum to determine the /

the standard for this disease. Then, in most instances, the normal of the patient was taken before I started the Horse Serum injections but their opsonic curves seemed practically unaffected by the remedy and I do not think any of my cases, (which I have detailed pages 120-134) derived appreciable benefit from this treatment.

During the nine months I spent in France as M'Cosh Scholar I devoted special attention to the subject of infant feeding and its relations to diarrhoea. I made elaborate analyses of the ingesta and excreta of infants and in this connection I have detailed the results of examination of twenty two specimens of human milk which I undertook to ascertain the cause of persistent and intractable diarrhoea in a number of breast fed infants which came under my notice. The estimations were conducted by a new method which I devised (pages 155-158). The results indicated that lactose is frequently present in human milk in greater quantity than has hitherto been regarded as normal and seemed to prove that its amount is an important factor in the causation /

ation of chronic diarrhoea in breast fed infants.

I also analysed 101 samples of the milk offered for sale throughout London in order to ascertain the average quality of that which formed the daily food of the infants suffering from diarrhoea and marasmus who were brought to the Sick Children's Hospital, Great Ormond Street, London, while I was resident there. My methods and results are fully described on pages 165-197.

I wish gratefully to acknowledge my indebtedness to the Faculty of Medicine of Edinburgh University who by their award of the M'Cosh Graduate's Scholarship gave me the opportunity of carrying on the greater part of this work.

C O N T E N T S.

C O N T E N T S

(Cont).

C O N T E N T S.

P L A T E S.



XII./

To face Page

## P L A T E S (Cont.)

# T A B L E S.



XIII/.

Tables Continued.

# C A S E S . - - -

| Case. No. | Name. | Termination D - death. C - cured | Duration of stay in hospital. |
|---|---|---|---|
| 1. | Charles Blackburn. | D. | 35 dys. |
| 2. | Kathleen McHenry. | D. | 27 " |
| 3. | Nellie Foxon. | D. | 6 " |
| 4. | Leo. Harris. | D. | 29 " |
| 5. | John Giovanni. | D. | 30 " |
| 6. | Gladys Pinchbeck. | C. | 35 " |
| 7. | William White. | C. | 21 " |
| 8. | Annie Fryer | C. | 10 " |
| 9. | Edward Dell. | C. | 8 " |
| 10. | Edward Kelsey. | D. | 37 " |
| 11. | Philip Bennett. | D. | 32 " |
| 12. | Florence Davies. | D. | 37. " |
| 13. | Gladys Brandon. | D. | 35 " |
| 14. | Charles Blackburn ? | D. | 35 " |
| 15. | Albert Quiver | D. | 8 " |
| 16. | John Farquhar. | D. | 7 " |
| 17 | Otto Linck. | D. | 8 " |
| 18. | Lilian Lindsay. | D. | 3 " |
| 19. | Beatrice Stiff. | D. | 36 hrs. |
| 20. | Violet Burrell. | D. | 15 dys. |
| 21. | Ethel Castle. | D. | 9 " |
| 22. | Frank Taberner. | D. | 7 " |
| 23. | Ivor Everard. | D. | 6 " |
| 24. | Florence Russell. | D. | 8 " |
| 25. | Eva Evans. | D. | 12. " |
| 26. | Marie Cameron. | D. | 21 " |
| 27. | Gladys Martinelli | D. | 2 " |
| 28. | Maud Lismore | D | 7 " |
| 29. | Ronald Sired. | D. | 4 " |
| 30. | John Mitchell. | C. | 14 " |
| 31. | Alfred Young. | D. | 7 " |
| 32. | William Harvett. | D. | 12 hrs. |
| 33. | Frank Marlow. | D. | 18 dys |
| 34. | Louisa Hunter. | D. | 1 mth |
| 35. | Arthur Fermon. | D. | 23 dys |
| 36. | Thomas Lee | D. | 7 " |
| 37. | George Smith. | D. | 24 hrs |
| 38. | Mary Palmer. | D. | 14 dys |
| 39. | James Bell. | D. | 3 " |
| 40. | William Roberts. | D. | 27 " |
| 41 / | | | |

contd.

| Case. No. | Name. | Termination D – death. C – cured. | Duration of stay in hospital |
|---|---|---|---|
| 41. | Sydney Hunt. | C. | 10 dys. |
| 42. | Rose Steadman. | D. | 20 mins |
| 43. | Dora Swartz. | D. | 13 dys. |
| 44. | Ida Jones. | D. | 14 " |
| 45. | Denise Walter. | D. | 5 " |
| 46. | Katherine Smart. | C. | 8 " |
| 47. | May Martin. | C. | 15 " |
| 48. | Richard Esmond. | D. | 4 " |
| 49. | John Skinner. | D. | 20 " |
| 50. | Cecil Butler. | D. | 5 " |
| 51. | Lucy Smith. | D. | 24 hrs. |
| 52. | Edith Walden. | D. | 4 dys |
| 53. | Florence Hosseman. | D. | 2 " |
| 54. | Sydney Strange. | D. | 3 " |
| 55. | Marjorie Walker. | D. | 30 mins |
| 56. | Amy Alzapied. | C. | 9 dys |
| 57. | George Stacey. | D. | 17 " |
| 58. | Dorothy Shears Relapse. | C | 14 " |
| | | D. | 12 hrs |
| 59. | Alfred Wood. | D. | 4 " |
| 60. | George Philpot. | D. | 12 " |
| 61. | Alice Best. | D. | 4 dys |
| 62. | Cecil Winkworth | D. | 6 " |
| 63. | Nelly Welsh. | D. | few hrs. |
| 64. | Rose Bradley. | D. | 12 " |
| 65. | William Newman. | D. | 6 dys |
| 66. | Florence Barnes. | D. | 30 hrs. |
| 67. | Alexander Laurentine. | D. | 3 dys |
| 68. | Ada Weyell. | D. | 5 " |
| 69. | James Ludham. | C. | 10 " |
| 70. | Rudolph Grover. | D. | 48 hrs |
| 71. | Ellen Hawkins. | D. | 6 dys. |
| 72. | Philip Lubrinsk. | D. | 24 hrs. |
| 73. | Leslie Dean. | D. | 1 hr. |
| 74. | Arthur Fox | Imp. | 9 days. |
| 75. | Kate Butler. | C. | 16 dys. |
| 76. | Fred Augur. | D. | 48 hrs. |
| 77. | Jessie Kendall. | D. | 12 " |
| 78. | Violet Harrison. | D. | 12 " |
| 79. | William Brooker. | D. | 12 " |
| 80. | Earnets Prior. | D. | 13 dys. |

# C H A R T S.

| Number | Charts. |
|---|---|
| 1 - 2 | Charles Blackburn |
| 3 - 4 | Kathleen McHenry |
| 5 | Nellie Foxon, |
| 6 - 7 | Leo Harris. |
| 8 - 9 | John Giovanni |
| 10-11 | Gladys Punchbeck |
| 12 | Annie Fryer |
| 13-14 | Edward Kelsey |
| 15-16 | Philip Bennett |
| 17-18 | Florence Davies |
| 19 | Albert Quiver |
| 20 | John Parker |
| 21 | Otto Linck |
| 22 | Violet Burrell |
| 23 | Ethel Castle |
| 24 | Francis Taberner |
| 25 | Ivor Everard |
| 26 | Florence Russell |
| 27 | Eva Evans |
| 28 | Marie Cameron |
| 29 | Gladys Martinelli |
| 30 | Ronald Sired |
| 31 | Arthur Fox |
| 32 | Ernest Prior |

## SYMPTOMATOLOGY.

To simplify my description of the cortege of symptoms which may appear in the course of this disease, I shall divide it into three:- 1st, the initial stage, 2nd. the acute stage, and third the subacute or marasmic stage.

1st. The initial stage:- Sometimes there seems to be a well marked period of onset which finds expression in restlessness and fretfulness. The appetite is diminished; the tongue is furred; and the motions are slightly more fluid and frequent than usual, but there may be no intestinal symptoms until comparatively late in the disease. In one of my cases, No. 3 , the onset was marked by grave disturbance of general health and a temperature of $103^{o}$, but there was neither diarrhoea nor vomiting till the fifth day. In my cases the acute condition was so often superadded to a chronic diarrhoea that the period between the onset and the appearance of the acute symptoms was usually ill defined. If we consider the period of onset as the stage in which the pathogenic organisms of this disease are either exalted in virulence or increased in numbers till they are able to produce the typical acute symptoms then its duration must vary according to

the organism. With very virulent organisms there will be no appreciable period of onset while with weakly organisms the period will be greatly prolonged. If through injudicious or pernicious feeding, a previous attack of diarrhoea, or any other cause the alimentary tract is unhealthy and little able to resist infection, then organisms normally of insignificant pathogenic activity, may acquire such an enhanced virulence as to produce diarrhoea and under these circumstances the preliminary undermining of the resistance of the alimentary tract may be looked upon as part of the initial stage of the acute infection. Sometimes, as in cases 43, 45 and 68, acute symptoms may appear with dramatic abruptness in infants enjoying apparently perfect health. An initial stage, therefore, although occasionally well defined, is an inconstant feature.

2nd Acute Stage:- The classic symptoms of the disease - diarrhoea, vomiting, and collapse - appear in this stage. Diarrhoea, anomalous as it may seem, is sometimes absent. I had not an instance of this among my cases but in many of them the diarrhoea was a subsidiary symptom. It varies in intensity within wide limits and is not therefore any index of the severity of the condition. Three

motions per day may be considered to be within the bounds of health. Greater frequency or increase in the bulk or fluidity of the motions, constitutes diarrhoea. In this disease the motions are usually frequent, copious and fluid; the linen may be constantly soiled. Their colour is mainly derived from the bile pigments, and the degree of hepatic reaction, thus manifested, is a very serviceable indication of the gravity of the infection. Usually they are yellow at first but the initial symptom may be the passage of a copious green stool. More frequently, however, as the disease progresses, the yellow passes through various intermediate shades till it merges into green and towards the end of the acute stage the motions may become colourless like water. The "rice water" stools of choleraic conditions in adults are very rarely met with although sometimes small white flakes are found in the motions which on examination prove to be epithelial cells and debris. The stools are not usually very foetid, but odours varying in intensity and repulsiveness are not uncommon. Their reaction is acid; sometimes so acid as to be intensely irritant to the skin; if, however, the previous food be sterilised milk they may be neutral or even alkaline. In five 1 of my cases the stools contained

blood, and then the presence of intussuception had to be carefully excluded. In chronic cases and in cases where the inflammation was seated mainly in the large intestine mucus was frequently observed in the stools.

Vomiting:- This was present in most of my cases. It was however absent in case 13 ; in Cases 6 and 10 - it was almost negligible; but in others, such as Cases 118 and 119 it was the predominant symptom and rapidly brought about the end. Its intensity, therefore, varied within wide limits and was no index to the severity of the condition. The vomited matter was frequently curdled, sour smelling partially digested milk but the product varied according to the nature of the food and the length of time which had elapsed since the preceding feed, Occasionally "retching" without vomiting was noted. Bile was rarely present in the vomited matter; there does not seem to be the same tendency for bile to pass back into the stomach of infants as of adults in severe vomiting. Haematemesis was noted in Cases- 20,.29 & 45.

Usually the vomiting and diarrhoea began about the same time. In a number of cases, however, the diarrhoea preceded the vomiting by a definite interval of several days - as in cases 7 & 39.

Vomiting was the initial symptom in cases 11. & 39.

Collapse:- This, the third of the cardinal symptoms, is invariably present. The degree is directly proportional to the severity of the intoxication. It may rarely, as in Case 3, precede the diarrhoea, it may be the only symptom of the condition but usually it is accompanied by either diarrhoea or vomiting or both. In fulminating cases it may be marked from the beginning but usually it is due to several factors; the toxaemia arising from the infection; the exhaustion caused by the vomiting and diarrhoea; by the absence of all absorption of nutritive material; and by the dessication of the tissues. These factors vary in importance in different cases. The main cause I think, is the toxaemia. Next, I would rank the withdrawal of fluid from the tissues upon which too little stress has been laid. I think thirst is the main source of suffering for these cases and the administration of plenty of fluid is a therapeutic measure which ought never to be neglected. The collapse may deepen into death or a temporary or permanent rally may take place.

The following is typical of the clinical picture presented in the acute stage:- The infant is very restless, fretful, and sleepless. Its arms,

head, and legs are in a state of constant unrest and every now and again it brusquely changes its position in bed as if vainly seeking for ease. Its cry is lusty and incessant at first, but gradually fades into a wavering whine. Sometimes, there are unappeaseable bouts of screaming as if the little patient was in an agony of pain, and terror; at others, the restless anxiety is replaced by a drowsy calm. Later, the movements become involuntary and unconscious; the eyes, lips, and facial muscles twitch; the fingers pick vaguely at the bed clothes; and clonic movements, sometimes with occasional tonic spasms of the limbs, may appear. Still later, the patient lies inert, flaccid, and motionless.

The facial expression is always anxious if the attack be at all severe . In bad cases the face is extremely pale and waxlike; the nose sharp; the skin wrinkled and fissured; dark rings appear round the eyes and gradually deepen till the eyes seem to sink into the depths of the orbit. Winking is infrequent or absent, the eyelids are not closed in sleep, and indent the margins of the exposed area of cornea.

The corneal surface ceases to glisten and ulcers appear on it; the ears are blue and cold; the lips are dry, thin, and cyanosed; the whole expression is one of intense suffering and anguish - a typical abdominal facies. The external temperature falls although a thermometer placed in the rectum usually registers a certain degree of fever. The limbs become cold and there is often a characteristic zone-like distribution of the algidity; e.g. the forehead may feel warm while the cheeks seem icy to the touch. The tongue is dry and furred; often brown and fissured; its margins are denuded of their epithelial covering. Thirst is always present and it is responsible for much of the infant's suffering as it renders suction painful and difficult.

ABDOMEN:- In the early stages it may or may not be tumid; the walls are resistant; the note is everywhere resonant; meteorism is very frequently met with. The intestinal movements seem to be accompanied by much pain in early stages but later they are painless as if the bowel had been anaesthetised; the abdominal wall rapidly becomes flaccid and folded and even deep palpation does not then

elicit any protest from the little patient.

Respiration:- Dyspnoea is usually present; the alae nasi are active and there is vigorous indrawing of the intercostal spaces. A varying degree of cyanosis appears in the extremities, lips, eyelids, nose and ears but it is often not well marked. The respiratory rate varies greatly but as a rule it is only slightly quickened; on an average it is from 30-40 per minute; the increased rapidity is readily explained by the elevated temperature; but each respiratory act is carried out with an intensity which seems to suggest an extreme thirst for air and raises a suspicion of a bulbar lesion.

Heart:- The heart rate is quickened, 120, 130, 140, 150, 160 beats per minute being not uncommon. Usually it varies between 120 and 140. The rhythm is regular, the beat is so feeble as rarely to produce a palpable impact on the chest wall. In only two of my cases were cardiac murmurs present, The sounds are feeble, and equidistant. As the collapse deepens the pulse becomes more and more thready till it is no longer perceptible at the wrist and temples and the exhausted heart becomes rapid, fluttering and irregular. Occasionally the

end is due to heart failure during a convulsive spasm.

Temperature:- I have already mentioned the dissociation of the peripheral and central temperatures. The chilling of the skin is one of the most salient features of the disease and Lesage alleges that the cold extremities and the zone of algidity localised to the cheeks is pathognomonic of Cholera infantum. The axillary temperature is rarely febrile. More usually it is seen steadily to decline from 98.4° to 97°, 96°, 95°, 94°, 93°, 92°, even to 91°. Similarly the buccal temperature is usually lowered, it varies between 97.5 and 98.6°. The rectal temperature is markedly febrile, as is seen in the accompanying Chart, No. 16 (John Parker). 101°, 102°, 103°, are not uncommonly registered. There is frequently a large daily excursion. But sometimes even in the acute stage the temperature is subnormal. In other cases, e.g. case No. 16 hyperpyrexia occurs. The high temperature is not usually sustained. Its persistence for more than ten days should raise a suspicion of typhoid or paratyphoid fever. There is one point I have specially noted regarding the temperature, viz. the

frequency of an antemortem rise. Out of my sixty-nine cases which proved fatal 18 showed this phenomenon. In some this elevation occurred in the last few hours; in others there was a continuous rise of temperature for 24, 48, and even 60 hours before death. The extent of this rise varies from 1.6° to 10.0°, and the average is 4.2°. The following table gives the details for each case:-

| Case No. | Name. | Ante Mortem Rise. | No. of Degrees. | Chart No. |
|---|---|---|---|---|
| 32. | Wm. Harnett | 100.8°-107° | 6.2° | |
| 36. | Thos. Lee | 98.0°- 99.8° | 1.8° | |
| 39. | Jas. Bell | 98.2°-100° | 1.8° | |
| 45. | Denise Walter | 97° - 104° | | |
| | No morning remission; rose steadily for 60 hours. | | 7.0° | |
| 48. | Edmonds, Rich. | 98.4°-102° | 3.6° | |
| 49. | Skinner, John | 98.4°-103° | 4.6° | |
| 50. | Butler, Cecil | 98.4°-100.4° | 2.0° | |
| 63. | Welsh, Nelly | 97.° -106° | 9.0° | |
| 65. | Newman, Wm. | 97° -101.8° | 4.8° | |
| 70. | Grover, Rudolph | 103.8°-106° | 2.2° | |
| 80. | Prior, Ernest | 98° -107° | 9.0° | |
| | Fell to 104° on sponging and did not rise again in the 4 hours which elapsed before death. | | | |

| Case No. | NAME. | Ante Mortem Rise. | No.of Degrees. | Chart No. |
|---|---|---|---|---|
| 32 | Taberner, Francis | 98.4°-102° | 3.6° | |
| 67. | Laurentum, Alex | 99.2°-105.8° | 6.6° | |
| 53. | Horseman, Flo. | 99.8°-101.4° | 1.6° | |
| 15. | Quiver, Albert | 100° -103° | 3.0° | |
| 16. | Parker, John | 97° -107° | 10.0° | |
| 56. | Alzapiedi, Amy | 98.4°-103.8° | 5.4° | |
| 60. | Philpot George | 98.4°-102° | 3.6° | |
| | Average of 18 cases | | 4.70° | |

Urine:- Normally an infant daily secretes from 10 to 17 ounces of urine during the first year.[1] It is clear yellow and has a specific gravity of 1002-1005. Owing to the great drain of fluid taking place from the bowel in diarrhoea, the amount of urine is greatly diminished; in severe cases it may be only two ounces or even one, per 24 hours. It is then deeply stained, turbid, acid and of a density of 1015 to 1020.

Normally, according to Lesne, the amount of urea per 24 hours varies between 0.30 and 0.50 grams per kilo. of the infant's body weight. Lesage states that the chlorides vary between 0.15 and 0.20 grams per 100 grams of urine. In summer diarrhoea both the urea and chlorides are diminished

1. Lesage Traite des maladies de l'enfance Paris 1904.

in proportion to the intensity of the attack.

The normal freezing point "$\Delta$" of infant's urine is from -0.20 to -0.40° C. According to the gravity of the condition it may be lowered to -1.00 or even to -1.60°C.

The ratio of the freezing point to the percentage amount of sodium chloride present in the urine is normally between 4 and 5.

$$\frac{\Delta}{NaCl} = 4 - 5$$

In acute infective diarrhoea it is considerably raised.[1]

$$\frac{\Delta}{NaCl} = 6\text{-}7\text{-}8 \text{ or even } 9.$$

The ratio of the nitrogen present as ammonia to the total nitrogen is also raised. The ratio of the nitrogen present as urea to the total nitrogen is lowered in acute cases owing to the hyperactivity of the hepatic cells. The urine in this affection proves very toxic on injection into animals. Glycosuria[2] is said sometimes to occur. Peptonuria and indicanuria[3] are frequent. Albuminuria is variable. Urobilinuria and the diazo reaction of Erhlich are met with only in cases of pulmonary complications.

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1. Carsiere et Monfet, Academie de Medicine, July 1897
2. Robin and Thiercelin, quoted by Lesage.
3. Lesage

MENINGISM: There is a series of nervous symptoms which frequently supervene either in the acute or chronic stage of this disease to which French writers have given the name meningisme. This pseudo meningitis may occur in many acute diarrhoeal states. It is characterised by extreme restlessness, convulsions, torpor, muscular rigidity or paralysis, and sometimes with tetany. Meningism was present in varying degree in many of my cases. I noticed it was usually a late symptom. In very acute cases algidity, coma and death follow one another so rapidly that the brain has no opportunity to react to the intoxication. Sometimes as in Case No. 31 , it is,however, present at the onset, and Concetti[1] describes cases arising during convalescence. The symptoms, I most commonly observed, were excitation; squint, ptosis, inequality of the pupils, stiffness of the neck, head retraction, and other localised muscular rigidities such as trismus and rigidity of arms and legs; brusque involuntary muscular contraction, and Cheyne-Stokes respiration.

The infective process having destroyed the protective power of the intestinal mucosa toxines pass into the blood and these nervous phenomena are partly symptomatic of the general intoxication

1. International medical Congress, Paris 1900.

and partly a reflex of the intestinal irritation. In only one, No. 15 of my 69 fatal cases was there any post mortem evidence of gross pathological change in the nervous system. In that case the meningitis was pneumococcal and its occurrence with diarrhoea was probably accidental. In cases of meningism the pulse is almost invariably rapid; the temperature is high; Kernig's sign is usually absent, and lumbar puncture (Cases No. 15, 16 & 27). shows an absence of leucocytosis.

Some fulminating cases perish within 48 hours, but the usual duration of the acute stage is from 3-8 days. The process may then go on to cure. Heat gradually returns to the cold extremities; the patient's distress becomes alleviated; the diarrhoea and vomiting cease, and the disease enters upon a period of convalescence. In other cases, life inappreciably merges into death. While others again pass into the third or subacute stage:- The onset of this stage may be delayed till about the fourteenth day; frequently it appears between the 8th and 12th. The virulence of the acute symptoms gradually diminishes; the vomiting practically ceases, the diarrhoea abates, and the patient /

patient enters upon a period of indefinite length during which the disease neither advances nor retrogresses. Intercurrent disease may intervene, most commonly pulmonary affections, to cut short the malady; but, usually after a certain resting period, without any marked alteration in the symptoms the patient seems perceptibly to waste away; he enters upon a condition of what I might term Acute Marasmus. Drugs, dieting, massage, hot baths, infusions, all seem powerless to arrest this fatal process and the case terminates in two to three weeks after the acute stage has passed. I have made special reference to the frequency of the stage in cases treated with "Lactated" milk, and I have under that heading made several suggestions as to its probable cause. Excluding my case of gaertner infection the following might be classed as dying in this third stage:-
Cases 10, 11, 12, 13, 26, 33, 35 and 49. Besides wasting there is another salient feature in this condition namely a tendency to sub-normal temperature. Evidently the condition is one of profound metabolic error for judging from the stools assimilation is being properly performed, but yet the infant is uanble to maintain its animal heat. The condition seems /

seems to be the result of the intense toxeamia which may accompany the acute stage. Definite nerve lesions such as occur as sequelae to diphtheria and other toxic conditions have frequently been described. Perhaps, this stage is but an indication of some such lesion, and many of these cases may thus be doomed from the very nature of the infection, no matter how successful the treatment of the acute stage may be.

I would like to call attention to a phenomenon I have frequently noted in this stage of the disease; viz, localised oedema. It occurs chiefly in the extremities, especially in the feet and legs. I often wonder how it arose but one of its causes was revealed in a most striking manner. The night before Case 49, John Skinner, died the lower half of the window at the head of his bed was inadvertently left open. He was warmly clad; well wrapped in blankets, and lay partially on his side so that the left side and the nasal portion of the right side of his face and the left side and part of the occipital region of his head were exposed to the cold night wind. I saw him two hours after he had been lying thus. Over all /

all the area exposed to cold a remarkable degree of oedema had developed. Its edge was sharply defined and its sinuous outline accurately represented the extent to which the child's head had sunk into the protecting pillow. After this case I had no more oedema for I always carefully safeguarded my little patients from cold.

# PATHOLOGY.

## P A T H O L O G Y.

Nearly all my cases were examined post mortem. The anatomical lesions in this disease vary with the acuteness of the attack.

In those which die within two or three days but little is found on post mortem examination:-

There is said to be a bluish hue of the intestines. The liver is normal or but slightly enlarged; and bleeds freely on section. The gall bladder is often distended with thick, acid green bile, an index of the intoxication. The bile is said to be sometimes blood stained[1]. The kidneys are congested; anuria is frequent; the other viscera are congested and cyanosed.

In cases of slower evolution a pallor replaces the congestion; the biliary function may be wholly suppressed and the gall bladder empty. Fatty degeneration is rarely met with. Amyloid degeneration has been described but the observation has not been confirmed. The reaction of the various abdominal viscera is said to be acid. Their anatomical structure shows practically no change.

Intestinal lesions:- The external surface is normal.

1. Lesage. Traite des maladies de l'enfance, Paris 1904

The mucous membrane of the stomach is thin and pale. Sometimes small patches of congestion, more rarely haemorrhagic areas, are observed on the summit of the mucous folds, on the villi and on the valvulae conniventes. This distribution on the most prominent parts of the mucous membrane, on the parts which come most intimately into contact with the intestinal contents, is most suggestive. Such lesions were present in several of my cases and I have noted them where they occurred. A variable amount of desquamation is usually evident. The degree depends upon the severity and duration of the attack. In cases of short duration, the mucous membrane is congested and oedematous. In cases of longer duration when the small intestine was mainly affected the large intestine was congested and oedematous and appeared to the naked eye to be the more seriously affected but on microscopical examination the small intestine was found to be extensively denuded of its epithelium.

Peyer's patches and the solitary glands were frequently observed to be enlarged although sometimes they appeared to be normal. This hypertrophy was observed in slight as well as in severe cases and seemed to depend more upon the lymphatic reaction of the case than on the intensity of the inflammatory process.

The changes in the epithelium are very variable. Heuber states that the epithelial cells may undergo mucoid degeneration, or necroses. Bagiusky places the main seat of the lesion in the glands and in the interglandular spaces rather than in the superficial epithelium. He describes swelling of the glandular cells, and hyaline, or mucoid degeneration; the latter process leads to the formation of the mucus found in the stools. The mucoid changes are more marked in the large intestine than in the small. There is often a marked small celled infiltration of the mucous membrane of the villi, glands, and of the inter-glandular spaces, and submucosa (see Plate IV.)

According to Holt the colon, lower ileum, and stomach suffer most; the duodeneum and jejuneum least. The cells with their nuclei degenerate; in ordinary cases mainly in the crypts of lieberkuhn: in more prolonged and severe cases the superficial epithelium is entirely destroyed and through the breach thus caused bacteria penetrate into the deeper parts of the intestinal wall.

Booker /

Booker states that superficial loss of the epithelium of stomach and gut is a constant lesion in all fatal cases of gastroenteritis. It is intact in some places, destroyed or eroded in others. The duodeneum and jejuneum may shew less denudation than other parts of the gut. The mucosa is infiltrated with mononuclear and polynuclear leucocytes. He also describes a deep ulceration affecting the crypts and villi.

Heubner has described a special form of necrosis in cholera infantum which chiefly affects the epithelial structure without involving the deeper layers of the mucosa.

No bacteria are found in the mucosa if the superficial epithelium is intact. The toxines elaborated in the gut first erode the superficial epithelium and pave the way for bacterial invasion. Organisms in the mucosa are more abundant in the superficial layers. In the deeper layers ( see plate) they are met with in nests of the inter glandular spaces. Marfan and Bernard[2] state that there is no relation between the presence of the bacteria in the wall and the Gravity of the disease.

Blood Vessels. The changes in them are variable. They may be distended and congested and small haemorrhages and exudations may be seen in their/

their neighbourhood. (See plate. IV.)

Liver:- The liver is congested in the acute stage: the capillaries are distended especially the intralobular and the hepatic cells are separated into groups of 2, 4, 6 or 10[3]. American authors have described fatty degeneration and necrosis. This change is denied by most French observers, but probably the Americans are dealing with quite a different pathological condition.. This explanation of the difference in the pathological descriptions is borne out by the preponderating influence of Shiga's Bacillus as a cause of summer diarrhoea in America.

Booker describes a necrosis of the epithelium of the convoluted and irregular tubules of the Kidney.

2. La Presse Medical 1899. p.289.

3. Terrien. These de Paris, 1894.

# BACTERIOLOGY.

## B A C T E R IO L O G Y.

In breast fed infants the motions for the first 20 hours of life are quite sterile. The normal flora of the intestine thereafter is subject to great variations within the limits of health. It varies in different localities of the intestine; in breast and bottle fed infants and in the latter according as they are fed with sterlised or non-sterilised milk[1]. Marked seasonal alterations are noted and geographical distribution seems also to be an important factor. Most diverse accounts of the bacteriology of this disease have been published by competent observers thus emphasing the fact that diarrhoea is but a common symptom of a number of allied diseases which we have only begun to differentiate.

In 1885 Escherich published a classic paper in which he divided diarrhoea into Ectogenous and Endogenus. He laid stress upon the power of the normal intestinal group of Bacillus Coli Communis to excercise a pathogenic action and he insisted upon the importance of an organism which he called the Streptococcus enteritidis in the causation of the Ectogenous variety /

variety. Booker[2] in 1886 corroborated much of Escherich's work; he stated that another organism normally found in healthy stools - Bacillus lactis aerogenes- could also acquire virulence and he found in many cases that the predominant organismal factor was Proteus vulgaris: (See also Escherich, Brud Zinski[3] and Ardorim[4]) and concludes that not one specific organism but many different varieties of bacteria are concerned in the etiology of summer diarrhoea. The causal relation of streptococci and diarrhoea has also been insisted upon by Flexner[5], Marfan[5a], Barbies[5b], and others[5c], and Cemiston has proved that the virulence of B. coli communis is exalted in presence of Streptococci[6]. Recent work at the Rockfeller Institute has further emphasised the importance of Streptococci especially in diarrhoea with green stools. This organism has been alleged to be frequently present in normal stools[7]. Kossel and Baginsky, Escherich, Nobecourt[8] and Blinn[9] have given prominence to this incidence of B. Pyocyaneus. Klein described an anaerobic organism which has a wide distribution and is found in milk and normal excreta. He isolated it from many cases of food poisoning which occured at St. Bartholomews Hospital in 1892 and called it the B. enteritidis/

enteritidis sporogenes. Many proteolytic bacteria also have been incriminated - Bacillus Subtilis ; B. mesentericus vulgatus[10]: and Tyrotrix tenuis, Delepine[11] in his report on the Manchester epidemic of 1894 concludes that infantile diarrhoea is caused by allied[12] organisms of the colon group, B. coli communis representing the one extreme and B. enteritidis of Gaertner the other. The most important, he decided, were those resembling B enteritidis of Gaertner

Lesage[13] in 40 out of 50 cases of infantile diarrhoea at Paris found the serum agglutinated B. Coli Communis. Gaffky[14], quoted by Newman, described 3 cases of acute diarrhoea caused by the use of milk from a cow suffering from an inflamed udder (garget) the causal organisms being B. coli communis and B. Gaertner he laid stress upon the dangers of foecal pollution of cow's milk.

A fresh impetus was given to the subject by the discovery of the B. dysenteriae by Shiga in 1898. Flexner[5] in Manilla, in 1900 confirmed Shiga's results and described a type which differs in certain cultural characteristics and agglutinative properties from the Shiga bacillus. Kruse[15] in Germany in 1901 described/

described what he claimed to be a new causal organism of dysentery but it is now regarded as identical with the Shiga Bacillus. In 1902 in a paper entitled "Etiology of Summer Diarrhoea in Infants" Duval and Bassett[16] published their results of the investigation of the causal relation between B. dyenteriae and summer diarrhoea in infants. The American type of acute infantile diarrhoea is characterised by blood and mucus in the stools. Shiga described the presence of blood and mucus in the stools as typical of the cases in which his organism was present. Duval and Bassett examined 53 suspected cases for the B. dysenteriae. They found it more abundant in the substance of the mucous membrane than in the intestinal contents.

53 cases examined.

42 B. dysenteriae present

11 B. dysenteriae not found.

Then they took 25 cases of diarrhoea, at random, and tested the agglutinative properties of their blood to B. dysenteriae, nineteen gave a positive reaction.

They tested 25 healthy infants in a similar manner/

manner but in none did they get any reaction to B. dysenteriae.

They concluded that B. dysenteriae "is an important, if not the most important cause of summer diarrhoea in infants."

Flexner[17] directed an investigation into the relationship of B. dysenteriae to summer diarrhoea in 1903. It was carried on simultaneously in a large number of hospitals and medical schools throughout the United States but many of the investigators were under-graduates. The results differed widely Flexner sums them up as follows:-

"The type of B. dysenteriae which preponderated in the children was the so called "Flexner Harris" organism. The Shiga type of the organism is exceptionally met with and occasionally both types are found in association ---Types of B. dysenteriae of less well established properties have also been encountered. The central fact brought out by this collective investigation is the frequent occurence in the diarrhoeal diseases of children of a specific micro organism which hitherto has been held to be of specific pathogenic action in human beings and to/

to be the cause of that form of dysentery among adults and also among children which is characterised by necrotic and pseudo membranous lesions of the intestine and marked infectiousness".

Morgan in 1905 undertook the bacteriological examinations of cases of acute infantile diarrhoea occuring in London. He says, "I determined to make a research into the bacteriology of as many cases of the disease in this country as I could collect during the time of prevalence last Summer. To this end it seemed advisable to collect all the aerobic bacteria of intestinal type present in the stools and intestines of these cases excluding however all the lactose fermenting and the gelatine liquefying bacteria, in order to determine which types were most prevalent. This was controlled by an examination of the stools of healthy children of similar age so that one might exclude from the list of possible causes any bacteria found constantly present in normal intestines". Out of 58 cases examined 49 were patients of Goldsmith Ward, Sick Children's Hospital, Great Ormond Street. The following table summarise his results :-

Table 1.- Morphological and Cultural Tests applied to the various Bacteria Isolated.

| No. | No. of cases in which found. | Morphology. | Glucose. | Mannite. | Dulcite. | Lactose. | Cane Sugar | Litmus Milk 1 day; | 3 days; | 15 days; |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 28 | B.Motile | A.G | – | – | – | – | 0 | 0 | Alkali |
| 2 | 3 | B. Non-motile | " | – | – | – | – | A | A | Alka. |
| 3 | 5 | " | A | A | – | – | – | A | A | 0 |
| 4 | 2 | " | A | A | – | – | – | A | A | 0 |
| 5 | 4 | B.Motile | A | – | – | – | – | A | 0 | Alks |
| 6 | 1 | " | A.G. | A.G. | A.G. | – | – | A | A.S | Alk. |
| 7 | 2 | " | A.G. | A.G. | A.G. | – | – | A.S | A. | Alk. |
| 8 | 1 | B. Non-Motile | A.G. | A.G. | A.G. | – | – | A. | 0. | A. |
| 9 | 1 | " | A.G. | A.G. | A.G. | – | – | A. | A. | Alk. |
| 10 | 4 | " | A.G. | A.G. | A.G. | – | A.G. | A. | A. | A.C. |
| 11 | 3 | B.Motile | A.G. | A.G. | A.G. | – | A.G. | A. | Alk. | Alk. |
| 12 | 2 | " | A.G. | A.G. | – | – | A.G. | A. | A. | A.C. |
| 13 | 1 | " | A.G. | A.G. | – | – | A.G. | A. | A. | A.C. |
| 14 | 5 | " | A.G. | A.G. | – | – | – | A. | A. | Alks. |
| 15 | 2 | Streptococci | A. | A. | A. | A. | A. | A.S. | A. | A. |
| 16 | 1 | " | A. | – | – | A. | A. | A. | A.C. | A.C. |
| 17 | 1 | Coccus | A. | – | – | – | – | 0. | A. | A.C. |
| 18 | 1 | " | A.G. | A.G. | – | – | – | A. | 0. | Alk. |

Table 1- Continued.

| No. | Litmus Whey 1 day | Litmus Whey 7 days. | Indol. | Sorbite. |
|---|---|---|---|---|
| 1 | Alks. | Alks. | ± + | |
| 2 | A. | " | − | |
| 3 | A. | A. | ± + | A. |
| 4 | A. | A. | − | − |
| 5 | A. | Alk. | − | |
| 6 | A. | Alk. | − | |
| 7 | A. | Alk. | ± | |
| 8 | A. | A. | − | |
| 9 | A. | Alk. | − | |
| 10 | A. | A. | + + | |
| 11 | A. | Alk. | ± | |
| 12 | A. | A. | − | |
| 13 | A. | A. | + | |
| 14 | A. | Alks. | + + | |
| 15 | A. | A. | − | |
| 16 | A. | A. | − | |
| 17 | O. | A. | − | |
| 18 | A. | Alk. | + | |

In order to compare the above table with the morphology and cultural reactions of some of the best known pathogenic intestinal bacteria, I add the following table compiled from "Some Observations upon the Micro-organisms of Meat Poisoning, and their Allies, British Medical Journal, June 10th, 1905, p. 1259).

TABLE II.

| Bacterium | Morph. | Glucose. | Mannite. | Dulcite. | Lactose | Cane Sugar | Litmus Milk 1 day | 3 days | 15 days |
|---|---|---|---|---|---|---|---|---|---|
| B. dysentry. Flexner Philippines | B.Motile | A. | A. | - | - | - | A. | Alks. | Alks. |
| B. dysentry Shiga... | " | A | - | - | - | - | A. | Alks. | Alks. |
| B. Typhoid. | B.Motile | A. | A. | - | - | - | A. | A. | A. |
| B.enteritidis Gaertner... | " | A.G. | A.G. | A.G. | - | - | A. | Alk. | Alk. |
| B.paratyphoid B,Schottmuller | " | A.G. | A.G. | A.G. | - | - | A. | Alk. | Alk. |
| B. paratyphoid A,Schottmuller | " | A.G. | A.G. | A.G. | - | - | A. | A. | A. |
| B. hog cholera, McFadyean | " | A.G. | - | - | - | - | A. | A. | A. |

A = Acid  G.= Gas.  C= Clot  S.= Slight.  - = No reaction.

| Litmus Whey. | | | |
|---|---|---|---|
| 1 day | 7 days | indol. | Sorbite. |
| A. | Alks. | ± | |
| A. | Alks. | - | |
| A. | A. | - | A |
| A. | Alk. | - | |
| A. | Alk. | Slight ÷ | |
| A. | A. | ÷ | |
| A. | A. | Slight ÷ | |

## TABLE III.

The various cultures are so arranged that those isolated by Dr. Morgan from infantile diarrhoea can be easily compared with known bacteria which, in some respects, resemble them

| Bacterium. | Motility. | Glucose. | Levalose. | Mannite. | Dulcite. | Maltose. | Dextrin. | Cane sugar | Lactose. | Galactose | Inulin. | Amygdalin | Salicin. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| No. 1 | B.Motile | A.G.S. | A.G.S. | – | – | – | – | – | – | A.G.S. | – | – | – |
| B. hog cholera, McFadyean. | " | A.G.S | A.G.S. | – | – | A.G.S. | A.G.S. | – | – | A.G. | – | – | – |
| No. 3 | B.Non " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| No. 4 | " | A. | A. | A. | – | – | – | – | – | A. | – | – | – |
| B.dysentery, Flexner, | " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| Phillippines. | " | | | | | | | | | | | | |
| B.dysentery, Gray | " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| B.dysentery, Strong | " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| B.from infantile dirrhoea | " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| Duval Baltimore | " | | | | | | | | | | | | |
| B.from infantile dirrhoea, | " | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| Duval, New York. | " | | | | | | | | | | | | |
| B.from monkey's faces, | " | A. | A. | A. | – | A.S. | – | – | – | A. | – | – | – |
| MacConkey 11. | " | | | | | | | | | | | | |
| B. typhoid. | B.Motile | A. | A. | A. | – | A. | A | – | – | A. | – | – | – |
| B. dysentery, Shiga | B Non " | A. | A. | – | – | – | A.S. | – | – | A. | – | – | – |
| B. dysentery, Kruse | " | A. | A. | – | – | – | A.S | – | – | A. | – | – | – |
| B.asylum dysentery, Eyre 7 | " | A. | A. | – | – | – | A.S | – | – | A. | – | – | – |
| B.asylum dysentery, Eyre 8 | " | A. | A. | – | – | – | – | – | – | A.S. | – | – | – |
| B.asylum dysentery, Eyre 9 | " | A. | A. | – | – | A.S. | A.S | – | – | A. | – | – | – |
| B.from monkey's faces | " | A. | A. | – | – | – | – | – | – | A. | – | – | – |
| MacConkey 1. | " | | | | | | | | | | | | |

T A B L E III (Cont).

| Arabinose | Raffinose | Sorbite | Erythrite | Indol. | Litmus Milk. 1 day | 3 days | 15 days |
|---|---|---|---|---|---|---|---|
| – | – | – | – | + + | 0 | 0 | Alk. |
| A.G. | – | – | – | + Slt. | A. | A. | A. |
| A. | – | A. | – | + + | A. | A. | 0. |
| A. | A.S. | – | – | – | A.S | 0. | Alks. |
| A. | A.S. | – | – | + | A. | 0. | Alks. |
| A. | A.S. | – | – | + | A. | 0. | Alks. |
| A | A.S. | – | – | + | A. | 0. | Alks. |
| A. | A.S. | – | A. | + | A. | 0. | Alks. |
| A. | A.S. | – | – | + | A. | 0. | Alks. |
| A.S. | A.S. | – | – | + | A. | 0. | Alks. |
| – | – | A. | – | +trace | A. | A. | Alks. |
| – | – | – | – | – | A. | 0. | Alks. |
| – | – | – | – | – | A. | 0. | Alks. |
| – | – | – | – | – | A. | 0. | Alks. |
| A.S. | – | – | – | – | 0. | 0. | Alks. |
| – | – | – | – | – | A. | 0. | Alks. |
| A.S. | – | – | – | – | A. | 0 | Alks. |

He concludes thus- These results, together with the evidence drawn from other sources, indicate that the bacillus No.1 is not an organism normally present in human stools, sewage, or in drinking water. From its prevalence in the stools and intestines of cases of infantile diarrhoea (in 28 cases out of 58), also from the fact that it causes death, preceded by diarrhoea in young animals, in the spleens of which it is invariably found in pure culture, one is led to conclude that this bacillus may possibly be a factor, or one of the factors, in the disease. A very significant fact is that the bacillus was isolated in pure culture(together with lactose-fermenting bacilli) from the stools of a nurse who had contracted diarrhoea from the patients in the ward set apart for this disease in the Hospital for Sick Children, Great Ormond Street.

The evidence in favour of bacillus No.3. being either the cause, or a cause of the disease, is perhaps almost equally strong, for although it was isolated in only 5 cases out of 58, one of these cases points very strongly to its being an infective agent, from the fact that the patient's blood agglutinated this bacillus, and also the typhoid bacillus. It was proved by experiment that this bacillus was agglutinated equally well by dysentery (Flexner) serum and by typhoid serum. That this patient's

serum/

serum, therefore, should agglutinate typhoid bacilli, as well as its own bacillus, is a very strong point in favour of this bacillus being infective in this particular patient at least. It proved pathogenic to young rats which were fed on it, although no diarrhoea was produced.

The evidence in favour of bacillus No.4. being a factor in the disease is based principally on its resemblance to the Flexner group of dysentery bacilli, which have been assigned as the cause of the disease in America. This bacillus, too, proved pathogenic to young rats when they were fed on it.

Bacillus No.1. and bacilli Nos.3 and 4 were found with about the same relative frequency in the cases clinically diagnosed as "acute infective diarrhoea" and "catarrhal diarrhoea." Bacillus No.1 was found in 11 out of the 28 cases of the former type, and in 17 out of the 30 cases of the latter type of the disease. Bacillus No.3. was found in two cases of "acute infective diarrhoea" and in three cases of "catarrhal diarrhoea", and bacillus No.4 in 1 of the former and 2 of the latter. It will thus be seen that the relationship between the clinical diagnosis and the bacteriology of the disease is not well marked. Dr. Batten informs me in/

in this respect that the distinction between these two types is not very definite, and it may very well be that the acute infective type is merely an aggravated form of the catarrhal.

The presence of a certain bacillus in the stools or in the intestines, in cases of disease, even although that bacillus be not found in the stools of healthy individuals, hardly proves it to be the cause of the disease. If, however, it has been found to invade the blood or the spleen, this evidence is very strongly in its favour. Unfortunately, neither the blood nor the spleen of fatal cases were examined bacteriologically, so that the presence or absence of bacteria in these localities are matters for future investigation by me next summer."

A bacteriological examination of the stools was made in four of my cases. Unfortunately, Dr. Le Morgan has not yet published the results, so I am able to state only the fact that he isolated one or two other of the above groups in cases 10, 12, 15, 16, 20, 22, 23, 25, 45, 46, 49, 52, 53, 54, 58, 64, 68, 71, 75 & 79.

I have recorded this in the description of each case. The cases seem to include all degrees of severity; some had green motions, others yellow: others/

others again colourless. In some the motions were frequent and foetid: in others the diarrhoea was slight. Two Nos.- 30 & 45, had Haematemesis. Case 45 had also blood in the stools. Cases Nos. 46 & 75 recovered: all the others died. From the scanty data at my disposal I cannot draw any conclusions excepting that the various organisms isolated by Morgan are capable of producing very different clinical pictures. Whenever he publishes his results, I shall see if these 21 cases can be divided into definite clinical groups each with a specific causal organism.

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## CLASSIFICATION.

The classification of diarrhoea is a matter of extreme difficulty. Bacteriology is slowly enabling us to differentiate them: in the first half of last century typhoid was separated: Gaertner in 1888 split off another group due to infection with the bacillus which now bears his name: a large group is composed of those arising from bacilli allied to Shiga's B' dysentericus: and another from bacilli allied to the B. coli communis. These groups have one great feature in common, the specific organism of each is agglutinated by the serum of the victim. As our knowledge advances differentiation by agglutination tests will replace the present unsatisfactory methods which are based on the clinical features of the case together with the appearance of the stools.

Batten[1] has described four varieties:-

1. Acute infective diarrhoea, the essential features of which I have just described.

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1. Clinical Journal January 3rd. 1906, p. 178.

2. Irritative diarrhoea, due to improper food or undigested food and characterised by loose stools, bulky, green in colour, with the presence of white curds and often with a sour odour.

3. Catarrhal diarrhoea, due to prolonged indigestion attended by the presence of mucus in the stools which are brownish green in colour and of foul odour.

4. Ulcerative colitis, a form attended with considerable pain and with the passage of mucus and blood, and is due to ulceration of the large intestine. It tends to run a prolonged course and is not accompanied by high fever.

These forms merge into one another. Patients often show symptoms belonging to two or more forms and I do not think the classification is of much practical importance.

A "dry" form has been described in which diarrhoea is either absent or slight. This was well marked in Case 3. of my series.

Escherich[1] has described a form in which from the /

1. Escherich Jahr fur Kinder, 1900 Vol. X1.

the beginning the symptoms are those of an acute intoxication: the patient is prostrated, the pulse is feeble and rapid, the diarrhoea is slight not foetid, yellow and alkaline: the abdomen is somewhat tumid; vomiting appears and with it all the general symptoms of cholera infantum.

Five cases were admitted for acute diarrhoea to my ward which were afterwards discovered to be cases of infection with Bacillus Enteritidis of Gaertner.

I shall now proceed fully to detail and discuss them.

# Cases of Infection

# with

# Bacillus Enteritidis of

# Gaertner.

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Out of 134 cases of diarrhoea which were in my ward of 12 beds during last summer, none gave a positive Widal reaction. Infections due to Shiga's Bacillus and to its Flexner variety were also absent. I had, under my care, however, three cases of diarrhoea due to Bacillus Enteritidis of Gaertner or rather one due to Bacillus Enteritidis of Gaertner, and two of a variety of it called Bacillus Aertrycke, which was isolated in a food poisoning epidemic in Germany and which is indistinguishable from B. Gaertner except for its agglutination reactions. Immediately before my entry to Great Ormond Street Hospital for Sick Children, there had been two other cases of B. Gaertner infection which, through the courtesy of Dr. F. E. Batten I am enabled to include. This makes a very interesting series of five cases of this infection. Two were cases of prolonged fever and diarrhoea in which I had repeatedly had the blood examined for the Widal reaction with negative results. My third case was of very short duration and had pyrexia diarrhoea and persistent vomiting. The pathological condition was thoroughly established in the case of Charles Blackburn by Dr. Graham Forbes, pathologist to the Hospital and he has kindly placed his notes at my disposal so that my record

and description of this condition might be as complete as possible.

Case [1] : Charles Blackburn, 1 year; he was a full term infant but had been "delicate" from birth. For the first three months of life he was breastfed and then two ounces of cow's milk and half an ounce of barley water were given two hourly.

His motions were always loose, 4-5 per day. He was first admitted to the Hospital for "wasting and convulsions" in February 1906, when he was aged 5 months, and was discharged much improved after 18 days. There were four other children alive in the family, but one was said to be "rickety" and "ailing"; 4 had died in infancy - 1 from diarrhoea, 1 from convulsions, 1 from broncho-pneumonia, and 1 from diarrhoea and vomiting.

He was admitted on October 1, 1906 suffering from diarrhoea of 10 days' duration. His motions were green, slimy, and frequent - 7-8 per day. The child was very collapsed - his anterior fontanelle was depressed, eyes sunken, pulse very feeble and extremities cold. He was emaciated and weighed only 11½ lbs. He had 4 teeth.

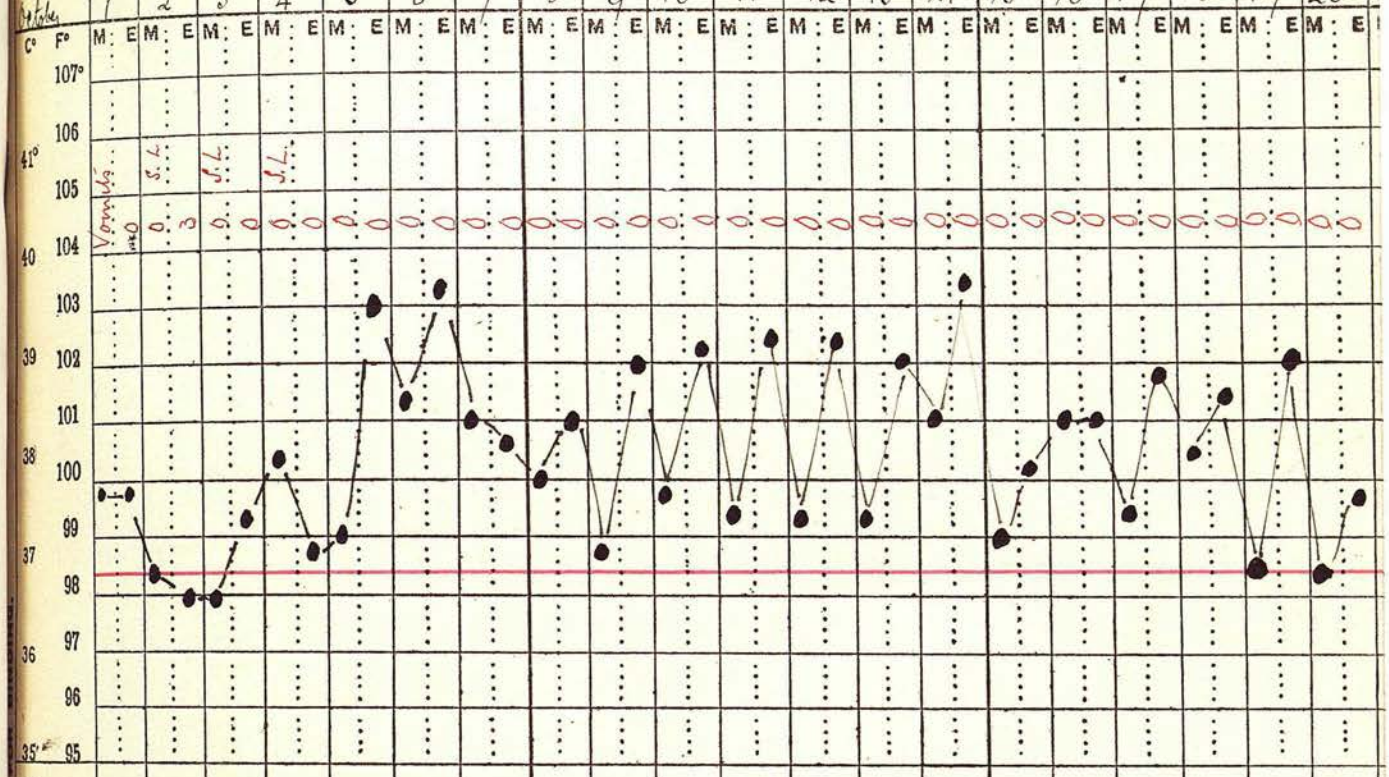
There was a slight strabismus present but no ptosis. The abdomen was distended but not resistant and everywhere tympanitic. The spleen

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 1 | Charles Blackburn | 1 year | Goldsmith | |

Disease

Result Death.

Date, October — 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20




| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 108 | 108 | 128 | 108 | 128 | 120 | 120 | 136 | 120 | 132 | 140 | 112 | 152 | 140 | 120 | 132 | 164 | 160 | 120 | 136 |
| Resp. | 32 | 36 | 32 | 36 | 40 | 36 | 44 | 40 | 48 | 36 | 28 | 52 | 60 | 40 | 40 | 48 | 36 | 44 | 52 | 32 |
| Bowels | 1 / 1 | 1 / 4 | 2 / 3 | 2 / 2 | 2 / 3 | 2 / 6 | 5 / 3 | 2 / 3 | 3 / 5 | 2 / 4 | 2 / 4 | 3 / 1 | 1 / 3 | 2 / 4 | 1 / 2 | 2 / 2 | 3 / 3 | 2 / 3 | 1 / 3 | 3 / 3 |
| Weight | | | lbs. oz. 11. 8 | | | | lbs. oz. 11. 0. | | | | | | lbs. oz. 10. 12 | | | lbs. oz. 11. 0 | | | lbs. oz. 11. 0 | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| Oct. 1st | Albumen Water. ℥ij<br>Brandy ♍ XX<br>2 hrly. | Oct. 1 Stomach and rectal lavage<br>Strychnine (1 in 400) daily.<br>Inj. hypo ♍ ij 3 hrly. for 6 doses.<br>Mustard Bath ~~p.r.n.~~ | | | |
| 2nd | Water<br>Lactated milk ā ā ℥j<br>2 hrly. | | | | |
| 6th | Lactated milk ℥ss<br>Peptonised milk ℥ss<br>Bengers ℥ss<br>Water ℥j<br>2 hrly. | 6th Resp. Stimul. for 4 doses. | | | |
| 10th | | 10th Mist: Carminative ℥j<br>Tinct: Nuc. Vom. ♍ IV | | | |
| 13th | | 13th Mist. Pot. Brom ℥½ } t.i.d.<br>Syp. Chlor. ℥½ } p.r.n.<br>Ward Salt. | | | |
| 15th | Peptonised milk ℥j<br>Bengers ℥j<br>Water ℥j<br>2 hrly. | | | | |

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 9 | Charles Blackburn | | Goldsmith & Alexandra | |

Disease

Result Death.

| Date, October | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | November 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 140 | 144 | 144 | 140 | 144 | 140 | 140 | 144 | 140 | 140 | 140 | 144 | 120 | 108 | |
| Resp. | 32 | 32 | 36 | 36 | 60 | 56 | 36 | 52 | 36 | 48 | 56 | 48 | 36 | 36 | |
| Bowels | 2 4 | 2 5 | 3 | 3 | 5 | 5 | 5 | 4 | 6 | 5 | 5 | 6 | 6 | 7 | 5 |
| Weight | lbs. oz. 10.12. | | | lbs. oz. 10.12. | | | | | | | lbs. oz. 9.12 | | | | |

Vomited 1 time

Transferred to Alexandra.

Stomach large & distended with gas

Abdomen very large

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

was slightly enlarged; the liver seemed normal. There were no adventitious sounds in the chest. The green slimy offensive nature of the motions suggested an acute diarrhoea supervening on a chronic. On the chart, No.1 the dietetic and medicinal treatment is fully detailed. For the first twenty four hours albumen water was given and various stimulants. He revived and next day "lactated" milk, 1 oz. and water 1 oz. two hourly were given. He improved so that three days later he seemed much stronger, the vomiting had ceased and the motions had entirely altered in character; they were now almost yellow, not slimy nor offensive but contained small flakes which on examination proved to be milk curds, so peptonised milk was given. The temperature began to rise five days after admission and continued high for three weeks - morning 99°, evening 102-103° (See Charts 1 and 2 ). On October 6th, a purpura developed on the abdomen and chest but persisted only a few days. From October 7-10, his general condition improved although he did not gain weight, nor did his temperature fall; the vomiting hadceased and the diarrhoea was very slight. On October 16th, twenty six days after the onset and sixteen days after admission, I

first tested for the Widal reaction, but got a negative result. Still seeking a cause for the persistent high temperature, I examined the ears but found nothing. On the advice of my visiting physician, I punctured them both but there was no pus; on October 19th, a slight serous oozing occurred from the left ear which on examination was found to contain cocci only. On October 19th, the blood was sent to Dr. William Marshall, one of my fellow students at Edinburgh University, of the Lister Institute. His report was:-

Gaertner.

Complete agglutination in ½ hour in dilution, 1-100. Normal Serum partial agglutination in ½ hour in dilution, 1-20 . None in dilution 1-50 or 1-100.

On October 20, there was profuse nasal discharge of which I had films and cultures taken, but no Gaertner Bacilli were found; it contained mainly cocci and a few bacilli which resembled the long and short forms of Klebs-Loeffler. The urine was offensive but contained no pus, albumen, nor blood. Gaertner's Bacillus was not found in it. The disease progressed uneventfully; and on October 24th

the temperature began to fall. My ward was then closed preparatory to re-opening as a ward for broncho-pneumonia so the case was transferred to the care of Dr. Garrod. There it continued to lose weight, the diarrhoea became more troublesome and the child steadily sank. It died on November 4th after having been under observation for 35 days.

Post mortem examination:-

Brain and thoracic organs - normal.

Liver and spleen seemed normal.

Stomach and duodeneum seemed normal.

Small ulcers began to appear a few inches below the duodeneum; they were placed irregularly at any part of the intestinal circumference; circular in shape; of a diameter about $\frac{1}{4}$ inch; of varying depths - one or two had reached the peritoneal coat; their edges were irregular; some appeared to have a minute slough; and there was one well marked area of ulceration with numerous ulcers about two inches above the ileo-caecal valve. There were altogether between 150 and 200 ulcers present. Peyers patches were swollen but not ulcerated. The large intestine was normal.

Microscopical:-

Heart - Slight fatty infiltration and of muscle fibres.

Lung - Many well marked areas of broncho-pneumonia; many alveoli plugged with cells and blood corpuscles; others emphysematous, distended, empty; alveolar capillaries congested.

Pleurae - thickened.

Liver - Much fatty infiltration chiefly of peripheary of lobule causing destruction and compression of numerous liver cells.

Spleen - Congested; capsule and trabeculae somewhat thickened - Malpighian corpuscles well defined.

Kidney - Cortex congested - Convoluted tubules show cloudy swelling and fatty charges in the epithelium. Many tubules contained hyaline debris and some glomeruli showed cell exudation under their capsule.

Mesenteric Gland - Congested - active lymph cell proliferation and areas of necrotic cells in the cortex.

Small Intestine - showed well marked ulceration with sloping margin extending to circular muscular coat which forms the base, underlying a layer of inflammatory cells which have also invaded the circular and longitudinal muscular coats extending through to the thickened peritoneum; the mucous coat for some distance on each side of the ulceration

is invaded and thickened by inflammatory cells and congested capillaries of the submucosa.

Bacteriological Examination:

The bacteriological report may be summarised thus:- The growth obtained was a Gram-negative motile bacillus. Pure cultures were got from the Spleen and Mesenteric glands; a mixed growth from the heart's blood.

The bile was sterile.

Subcultures were made on a series of media and the organisms gave the characteristic reaction of the B. Enteritidis of Gaertner. Experiments on animals were kindly carried out by Dr. Klein and Dr. T. Musfield, and confirmed the above observations. Dr. Klein supplied the blood serum of a rabbit rendered immune to Gaertner's B. and a number of agglutination tests were applied to the original cultures. They showed partial agglutination in dilution of 1 in 50 in 20 minutes, and complete in 1 hour.

| Tests. | Organism B.Motile Gram negative. | Agar Culture Greyish opalescence | Glucose Gelatine Shake Abundant Gas formation. | Gelatine Shake Slight gas formation. | Litmus Milk Litmus Acidified Milk not clotted. | Neutral Red Fluorescence . | Conradi Plate. Growth of blue colonies | Broth no Indol formed. | Agglutination. Serum of Guinea pig immune to Gaertner; marked 1 in 25 dil. |
|---|---|---|---|---|---|---|---|---|---|
| Blood | | | | | | | | | |
| Spleen | " | " | " | " | " and becoming grey blue afterwards. | " | " | " | " |
| Gland | " | " | " | " | Litmus acidified milk not curdled | . Fluor. | —+— | " | " |

Guinea pig fed on cultures from the blood died in 4 days.
Nothing definite found post mortem. Cultures from heart's blood gave:-

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Guinea Pig's Heart blood. | " | " | " | " | " | " | —+— | Trace of Indol. | " |

Cultures from spleen and peritoneal fluid sterile.

Dr. Klein:- "I have made subcultures of your microbe and find it behaves like Gaertner."

"Death due to general infection from a bacillus indistinguishable from Gaertner".

T. G. FORBES.

Case 2 , Kathleen McHenry, 1 year; admitted July 16th 1906 with diarrhoea and vomiting of one week's duration. Child was a full term child of a normal labour. For the first nine months it was breastfed; during the past three months it has had bread and milk at a crèche during the day with breast feeds morning and night. Only other child of family is aged three years and quite well.

On admission she was very collapsed and brandy and strychnine were freely exhibited. The temperature was 100°; she was very restless, screaming incessantly when awake; and during her short intervals of sleep, her facial muscles were seen to twitch. She was given albumen water and during the first twenty four hours she vomited thrice and her bowels moved eight times. Physical examination proved negative. She rallied; the diarrhoea became less and in two days the vomiting had ceased. As the temperature continued high, the suspicion of typhoid arose but the Widal reaction proved negative on two occasions; first on July 24. after she had been 9 days in hospital, and again on July 31, on the 16th day in hospital and the 22nd of the disease. The pulse and respiration increased in rate. On August 7th, a few crepitations could be heard over both bases; the neck was then observed

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 3 | Kathleen Mc Henry | 14 years | Goldsmith | |

Disease

Result Death.

| Date | July 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | August 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 160 | 180 | 128 | 124 | 128 | 124 | 140 | 160 | 168 | 172 | 122 | 176 | 140 | 160 | 160 | 160 | 160 | 184 | 176 | 164 |
| Resp. | 148 | 36 | 42 | 44 | 52 | 48 | 48 | 76 | 72 | 40 | 48 | 92 | 72 | 40 | 52 | 56 | 56 | 56 | 68 | 60 |
| Bowels | 2 | 6 3 | 4 3 | 4 2 | 2 1 | 1 1 | 3 2 | 2 2 | 3 3 | 4 4 | 3 3 | 3 2 | 4 3 | 3 4 | 3 4 | 4 5- | 4 3 | 5- 3 | 5- 3 | 4 4 |
| Weight | | | | lbs 14 | | | | lbs. oz. 14.4 | | | | | | | lbs. oz. 13.4 | | | | | |

| Date | Diet | Treatment |
|---|---|---|
| July 16 | Albumen Water ℥ ii<br>Brandy ♍ xx<br>2 hrly. | July 16<br>Stomach and rectal lavage. 31<br>Gl. Ricini<br>Inj. hyp. Strych. (1 in 100) ♍ ii<br>Statim |
| 24 | Do. + whey ℥ i<br>2 hrly. | July 17<br>Saline infusion ℥ vi<br>Resp. Stomach lavage. |
| 26 | Alb. Water ℥ ii<br>Whey ℥ ½<br>2 hrly. | ℞ Tinct. Opii B.P. ♍ ¼<br>Mist: Carminativa ℥ i<br>4 hrly. |
| 31 | Peptonised milk ℥ ½<br>Whey ℥ ½<br>hrly. | July 18.<br>Omit Opium. |
| August 6 | Citrated Melk ℥ ½<br>Lime Water ℥ ½<br>hrly. | July 19 Rectal lavage.<br>Mist: Carminativa ℥ i |
| 9 | Whey ℥ i<br>hrly. | July 20 Mist: Pot. Brom ℥ ½<br>4 hrly. |
| 10 | Whey ℥ ii<br>hrly. | |

| Date | Diet | Treatment |
|---|---|---|
| Aug. 11 | Whey ℥ iii<br>Brandy ♍ xv<br>2 hrly. | July 20. 11 p.m.<br>Mist: Pot. Brom ℥ ½<br>July 21.<br>Inj. Hydrar. Ox. Flav. para oculis.<br>July 23.<br>Resp. Stomach lavage.<br>July 25<br>Mist: Pot. Brom ℥ ½<br>p.r.n.<br>July 26<br>Rectal lavage.<br>July 28<br>Pot: Brom. grs 1½<br>4 hrly.<br>Rectal lavage.<br>Inj. Protargol 0.5% per rectum |

MARGIN TO BE LEFT FOR BINDING.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 4 | Kathleen Mc Henry. | 1 year | Goldsmith | |

Disease

Result Death

| Date August | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|---|---|---|---|---|---|---|---|
| Pulse | 160 | 184 | 180 | 160 | 184 | 140 | 176 |
| Resp. | 84 | 60 | 64 | 52 | 48 | 64 | 80 |
| Bowels | 3 / 3 | 3 / 1 | 3 / 5 | 6 / 4 | 5 / 4 | 5 / 2 | 5 / |
| Weight | | lbs. oz. 13. 4 | | | | | |

Vomits. 0 1 0 0 0 0 0 0 0 0

| Date | Diet | Treatment |
|---|---|---|
| | | Aug. 2. Tinct. Opii m½ Mist. Camphorata 3i 4 hrly. |
| | | Aug. 4 Inj. Strych. hyp. (1 in 400) mii 4 hrly. Brandy Tinct. Opii |
| | | Aug. 7. Protargol grs ⅛ Water 3i by mouth 3i 2 hrly |
| | | Aug. 8. Tepid sponge 3 hrly. If temperature exceeds 103°F. |
| | | Aug. 9. Alkaline lotion for nose |
| | | Aug. 10 Rectal lavage. |

| Date | Diet | Treatment |
|---|---|---|
| | | |

MARGIN TO BE LEFT FOR BINDING.

to be stiff; there was slight squint, and a fine tremor in the arms. The optic discs I found normal and the case was now supposed to be one of tubercular meningitis. On August 8, a faint purpuric, petechial rash was seen on the abdomen. On August 9th, the rash was spreading; the child was very restless; the stools were green and cheesy; the spleen was palpable; I could obtain only the right knee jerk; the left foot was rigid and flexed, but there was no Kernig's sign. On August 10th, the temperature rose to 103.8°; fine tremors were seen in the legs. On August 11th, the rash was fading; ptosis of the right eyelid appeared; the head retraction became much more marked; the pulse became more rapid and very feeble; then irregular; and the patient died.(See Charts 3 and 4).

Post mortem Examination -

Brain, lungs, and heart appeared normal.

Liver was enlarged and fatty.

Spleen was slightly enlarged.

Over the stomach mucosa for about half an inch there was a white membrane which could for most part be peeled off; in some places it seemed more adherent and the mucosa there appeared congested to the naked eye.

The intestines seemed perfectly normal; no sign of inflammation of Peyer's patches was present. The Mesenteric Glands were large and soft.

Microscopical:-

Liver - fatty infiltration.

Bacteriological:- From pure cultures of the spleen and mesenteric glands the Bacillus Aertrycke was isolated.

Case 3 , Nellie Foxon, 6/12; this was a most interesting case for it seemed to have a preliminary acute stage without diarrhoea. She was very ill for 3 days before she came under our notice. The mother stated that about July 29 the child became "queer" and had difficulty in swallowing . On August 2, she was brought to us, acutely ill with a temperature of 103°. We could find absolutely no cause for the fever and were in doubt whether or not we were dealing with deep seated pneumonia for the respirations were somewhat quickened also. On August 4th, diarrhoea and vomiting suddenly began. The motions were first green and then colourless; the vomiting was very

| No. 5 | NAME Nellie Foxon | AGE 6/12 | WARD | UNDER CARE OF |
|---|---|---|---|---|
| DISEASE | | | | RESULT Death |

DATE August 6 7 8 9 10 11

| Co | Fo | 6 M | 6 E | 7 M | 7 E | 8 M | 8 E | 9 M | 9 E | 10 M | 10 E | 11 M | 11 E |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 107° | | | | | | | | | | | | |
| | 106 | | | | | | | | | | | | |
| 41° | 105 | Vomits 6. | | 8 | | 6 | | 1 | | 7 | | 2 | |
| | 104 | | | | | | | | | | | | |
| 40 | 103 | | | | | | | | | | | | Saline infusion 3vj |
| | 102 | | | | | | | | | | | | |
| 39 | 101 | | | | | | | | | | | | |
| | 100 | | | | | | | | | | | | |
| 38 | 99 | | | | | | | | | | | | |
| | 98 | | | | | | | | | | | | |
| 37 | 97 | | | | | | | | | | | | |
| | 96 | | | | | | | | | | | | |
| 36 | 95 | | | | | | | | | | | | |
| 35 | | | | | | | | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| ~~PULSE~~ RESP | 64 | 50 | 44 | 46 | 50 | 60 2 hrs 80 |
| ~~RESP.~~ PULSE | 160 | 120 | 126 | 130 | 120 | 126 |
| BOWELS | 2 | 3 / 3 | 3 / 3 | 5 / 5 | 3 / 3 | 4 |
| WEIGHT | | | | | | |

| DATE | DIET | TREATMENT | DATE | DIET | TREATMENT |
|---|---|---|---|---|---|
| | | | | | |

MARGIN TO BE LEFT FOR BINDING.

frequent. The child had previously been always healthy. On admission, August 6th, the patient was somewhat collapsed, constantly screaming and very restless. There were no eye symptoms; the lungs were clear; the heart was feeble but regular; the abdominal wall was somewhat resistant; neither spleen nor liver could be felt; and the knee jerks were present. In spite of all treatment, she rapidly sank. On August 10 the fontanelle was observed to be more depressed; the knee jerks could no longer be elicited; she slept with her eyes only partially closed. The left pupil was seen to be larger than the right. On August 11, a corneal ulcer appeared on the right eye; a rectal examination proved negative; occasional convulsive movements of the limbs were seen and now and again a fish like movement of the lower jaw. The temperature rapidly rose to 105.2°; the heart became very rapid - 170 per minute; the respiratory rate rose to 80 and the patient died. There had never been any pulmonary signs and there never was any blood in the stools. The post mortem examination revealed nothing - no areas of congestion in the intestine - no ulceration - and only slight enlargement of Peyer's patches and the solitary glands. The spleen and mesenteric glands gave cultures of

the Bacillus Aertrycke. (See Chart 5)

For the notes of the two following cases I am indebted to Dr. F. E. Batten for they occurred just before I took charge of the ward.

Case 4 , Leo Harris, 4/12; was admitted for chronic diarrhoea on June 18th. His motions were usually greenish white;, for a few days they had been green and slimy; he was vomiting occasionally; he screamed and cried incessantly.

He was a first child and had never been breast fed, said to have been premature — 7 months gestation. On May 28th he weighed 7 lbs.10 oz; his motions were slate coloured and he was vomiting frequently. On June 11th he weighed 7 lbs. His diet had till then been cow's milk and barley water; it was now changed to "citrated" milk. On June 18 he weighed 6 lbs.11 oz. and was put on Nestle's milk.

On admission he was very weak and emaciated. His lungs were clear; there was a soft blowing systotic mitral murmur . The liver was enlarged and firm and its edge was sharply defined. The spleen was not felt. From his charts No.6 & 7 it

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 6 | Leo Harris | 4/12 | | |

Disease

Result Death.

| Date | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Time | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E |
| Vomits | 3 | 2 | 2 | 0 | 1 | 1 | 5 | 3 | 2 | 1 | 2 | 2 | 2 | 0 | 1 | 2 | 1 | 2 | 3 | 4 |
| Pulse | 132 | 130 | 136 | 124 | 130 | 120 | 132 | 124 | 136 | 140 | 132 | 135 | 126 | 130 | 122 | 136 | 124 | 120 | 126 | 140 |
| Resp. | 36 | 32 | 30 | 32 | 30 | 34 | 38 | 24 | 26 | 24 | 24 | 22 | 26 | 28 | 36 | 32 | 38 | 40 | 32 | 42 |
| Bowels | 0 / 0 | 2 / 2 | 3 / 3 | 2 / 3 | 3 / 3 | 3 / 4 | 3 / 4 | 3 / 2 | 3 / 2 | 4 / 7 | 4 / 3 | 4 / 3 | 2 / 3 | 3 / 3 | 2 / 2 | 1 / 5 | 3 / 4 | 2 / 4 | 2 / 2 | 2 / 3 |
| Weight | 7lb 4 | | | | | | | 7lb 6 | | | | | | | | 7lb 10 | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

MARGIN TO BE LEFT FOR BINDING.

| No. 4 | Name Les Harris | Age 4/12 | Ward | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| Date July – | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|
| M | | | | | | | | | |
| E | 3 | 4 | 5 | 3 | 0 | 3 | 0 | 1 | 0 |
| Pulse | 130 | 136 | 148 | 140 | 154 | 138 | 130 | 142 | 132 |
| Resp. | 36 | 38 | 46 | 42 | 48 | 42 | 40 | 46 | 48 |
| Bowels | 3/3 | 2/3 | 2/3 | 3/0 | 4/4 | 2/2 | 1/3 | 0/3 | 2/ |
| Weight | | | | | 7lb 8 | | | | |

Vomit 3v 3iv 3iv 3iv 3iv 3iv 3iii

Saline injection 4 " " 4

94°2

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

MARGIN TO BE LEFT FOR BINDING.

is seen that throughout the first three weeks of his illness there was a considerable daily range of temperature. In the fourth week there were some remarkable variations, a temperature of 94.2° being recorded five days before the child died. At first the child gained steadily but slowly in weight; the motions remained loose, frequent, and undigested. He was fed first on cow's milk 1 oz. and barley water 1 oz. and then raw meat juice in drachm doses was added to his feeds. But from July 7th he rapidly sank and died on July 16th.

On post mortem examination, a slight roughening of the mitral valve was found; the liver and spleen seemed healthy; the Mesenteric glands were somewhat enlarged. There was slight enlargement of Peyer's patches and the solitary glands. There were no areas of ulceration nor congestion.

Bacillus Enteritidis of Gaertner was isolated from spleen and mesenteric glands in almost pure culture.

Case 5 , John Giovanni, 3/52; no vomiting; diarrhoea since birth.

This was a full term child of a normal labour,

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 8 | John Giovanni | 3/52. | | |

Disease

Result Death.

| Date | June 29 | 30 | July 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Temperature M. | Vomits 0. | 2. | 3 | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 3 | 1 | 1 | 2 | 2 | 0 | 1 | 1 |
| Temperature E. | | | | | | | | | | | | | | | | | | | | |
| Pulse | 110 | 112 | 108 | 114 | 120 | 112 | 120 | 116 | 108 | 110 | 120 | 112 | 120 | 160 | 140 | 112 | 112 | 120 | 128 | 124 |
| Resp. | 32 | 30 | 32 | 36 | 32 | 32 | 36 | 38 | 40 | 30 | 36 | 36 | 44 | 60 | 48 | 40 | 68 | 40 | 64 | 72 |
| Bowels | 1 | 2/1 | 0/1 | 1/1 | 1/3 | 2/2 | 2/3 | 2/3 | 3/3 | 2/3 | 1/3 | 2/3 | 2/3 | 3/4 | 2/3 | 3/1 | 3/3 | 3/3 | 3/3 | 3/5 |
| Weight | | | | 3 lbs 8 | | | | | | | | | | 3 lbs 11. | | | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

MARGIN TO BE LEFT FOR BINDING.

| No. 9 | Name John Giovanni | Age 3/52 | Ward | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result |

| Date July – | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| C° F° | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E | M: E |
| 107° | | | | | | | | | | | | | | | | | | | | |
| 106 | | | | | | | | | | | | | | | | | | | | |
| 41° 105 | | | | | | | | | | | | | | | | | | | | |
| 104 | | | | | | | | | | | | | | | | | | | | |
| 40 103 | | | | | | | | | | | | | | | | | | | | |
| 102 | | | | | | | | | | | | | | | | | | | | |
| 39 101 | | | | | | | | | | | | | | | | | | | | |
| 100 | | | | | | | | | | | | | | | | | | | | |
| 38 99 | | | | | | | | | | | | | | | | | | | | |
| 98 | | | | | | | | | | | | | | | | | | | | |
| 37 97 | | | | | | | | | | | | | | | | | | | | |
| 96 | | | | | | | | | | | | | | | | | | | | |
| 36 95 | | | | | | | | | | | | | | | | | | | | |
| Pulse | 130 | 120 | 128 | 120 | 120 | 128 | 120 | 144 | 128 | 126 | | | | | | | | | | |
| Resp. | 44 | 76 | 48 | 40 | 36 | 64 | 40 | 52 | 64 | 50 | | | | | | | | | | |
| Bowels | 3/5 | 3/5 | 5/7 | 2/5 | 4/4 | 2/5 | 3/5 | 3/4 | 3/4 | 5 | | | | | | | | | | |
| Weight | | | | | | | | | | | | | | | | | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

MARGIN TO BE LEFT FOR BINDING.

but weighed only 3 lbs. at birth. It was spoon fed - cows milk 2 dr. water 1 dr. and brandy a few drops frequently. This was the eighth child and a miscarriage had occurred just before its conception. The immediately preceding infant was 3 years old and quite healthy. On admission he was very puny weighing only 3 lbs.8 oz.; he seemed always asleep, and never cried. Nothing was found in his chest or abdomen. His temperature was irregular and febrile (see Charts 8 & 9). His motions were loose and green; at first they were only two or three per day, but later they became more frequent, 6-8 per day. There was occasional vomiting. He improved for a short time; he gained a few ounces in weight with great regularity every week, till he reached 4 lbs. 3 ozs. From July 20th his motions were markedly loose; he became gradually feebler; he never voluntarily moved and slowly this lethargic state merged into death. On post mortem examination the brain, lungs, heart, liver, kidney, and spleen seemed normal to the naked eye, so no microscopical examination was made. There was no congestion nor ulceration in the intestine. Cultures from the spleen and mesenteric glands gave a growth which was indistinguishable from Bacillus Enteritidis of Gaertner.

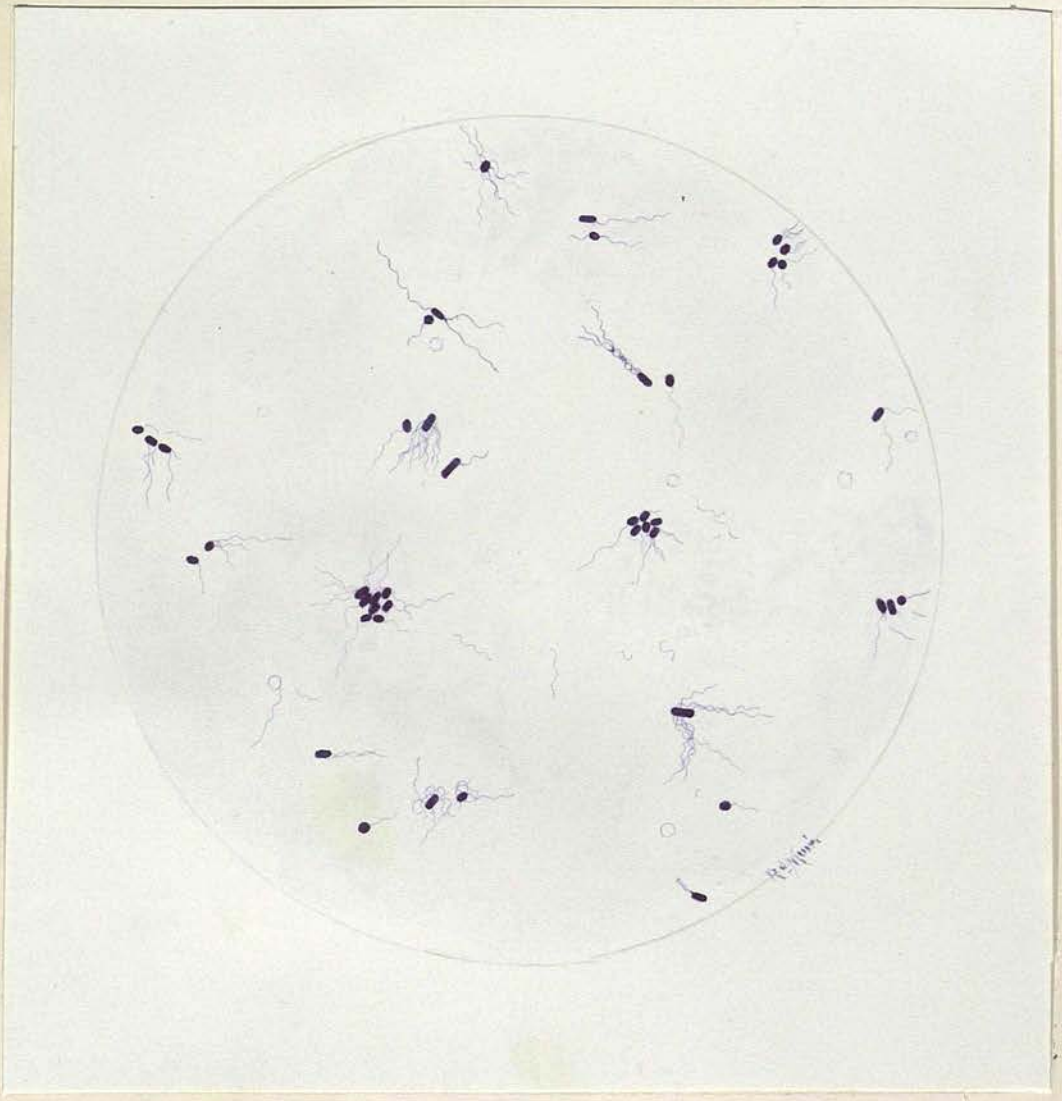


PLATE I

All my five cases died; the average duration of their stay in hospital was 25 days; the shortest was 6 days and the longest 35. Excluding the one acute case No. 3 which died in 6 days the average stay in hospital was 30 days.

| Case No. | days under observation | days ill before admission. | Duration of illness. |
|---|---|---|---|
| 1. | 35. | 10 | 45 |
| 2 | 27 | 7 | 34 |
| 3 | 6 | 3 | 9 |
| 4 | 29 | ? | ? |
| 5 | 30 | ? | ? |

There seems to be a definite acute stage which may end fatally from exhaustion or may pass into a chronic condition which tends to last from 3 to 4 weeks. In other instances such as cases 3 & 4 the disease seems to run a subacute course throughout. Temperature ;- In all there was a marked tendency to prolonged fever which was subject to considerable daily variations. Case 1 had a maximum temperature of 103.4°, a minimum of 97° and an average of about 99.2° : Case 2 max. 103.6°; min. 99° ; average 101° Case 3 max. 105.4°; Min. 98°; average 101.8°: Case 4 max. 102°, min. 94.2° ; average 98.6; Case 5, max. 101.2° , min. 96.6° ; average 98.4. The chronic cases had a greater tendency to intermittent subnormal temperatures. The pulse and respiration were increased /

PLATE II

increased in rate.

| case No. | Pulse. | | | Respiration. | | |
|---|---|---|---|---|---|---|
| | Max. | Min. | Average. | Max. | Min. | Average. |
| 1 | 160 | 108 | 135 | 60 | 28 | 40 |
| 2 | 184 | 122 | 165 | 84 | 36 | 60 |
| 3 | 172 | 120 | 140 | 80 | 44 | 55 |
| 4 | 154 | 120 | 135 | 48 | 22 | 36 |
| 5 | 160 | 108 | 125 | 72 | 30 | 44 |

The average of the pulse rate is seen to have varied from 125 to 140 and of the respiration rate from 40 to 60 per min.

I have already discussed the pathology and bacteriology of the condition. Plates 1(1X 1000). taken from a young culture; it shows well the lateral and terminal flagella and also depicts the curve assumed by them when they are shed. Plate 2 shows the shelving margin of an ulcer from the intestine of case 1. There is a large celled in filtration extending through the muscular coats causing atrophy and destruction of the muscle and thickening of the peritoneal muscle and thickening of the peritoneal coat. Plate 3 is a section of the stomach wall from case 2. It shows a fibrinous exudate on the surface /

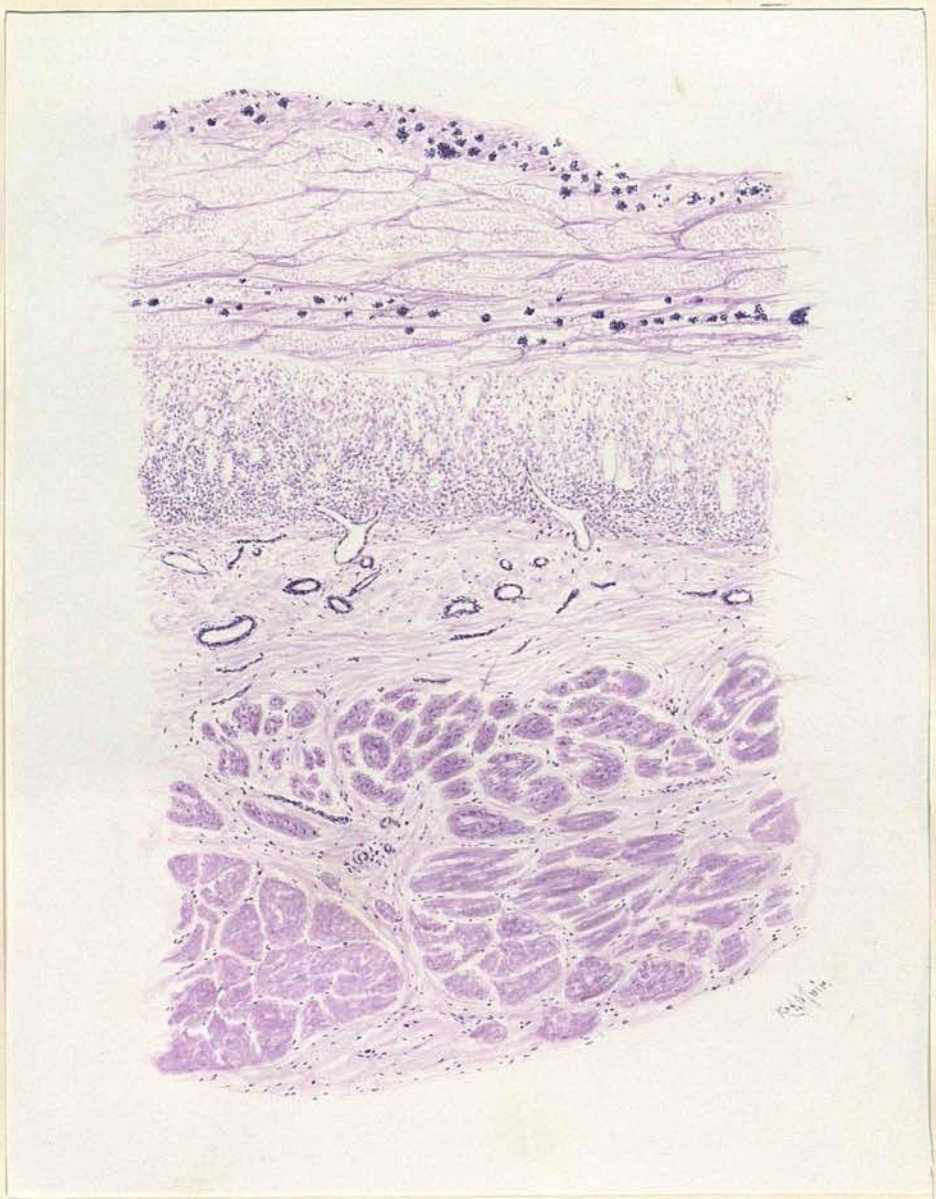


PLATE III

surface of the mucous membrane which contains masses of B. enteritidis of Gaertner. The mucosa also shows plugs of these organisms.

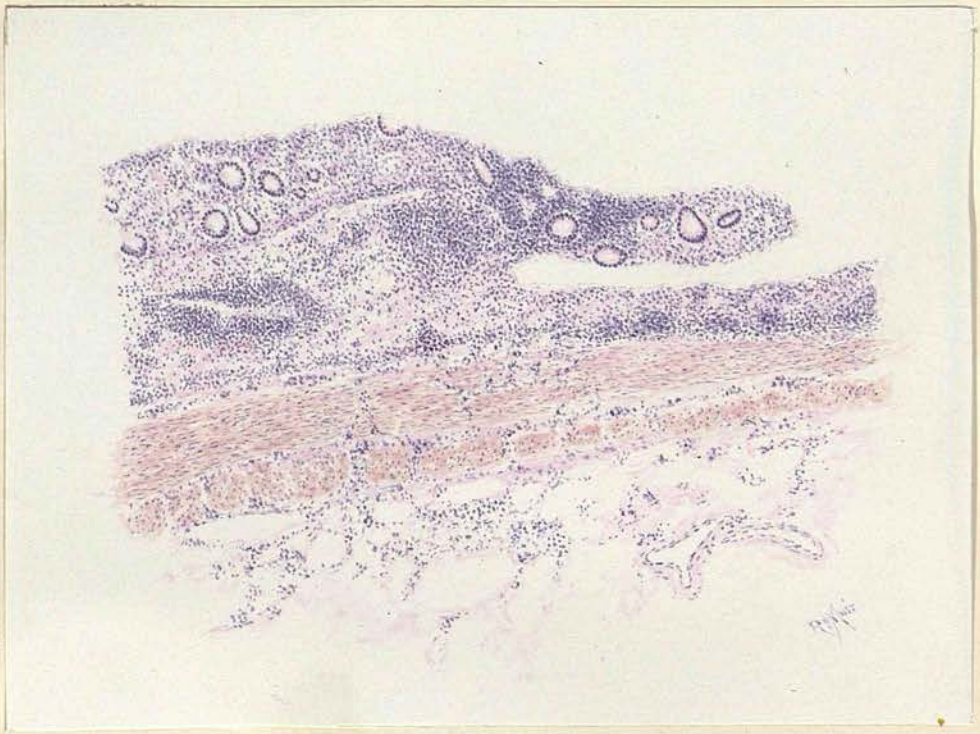


PLATE IV

# TREATMENT.

## T R E A T M E N T.

The treatment of cases of acute infective diarrhoea is often a matter of much difficulty for it entails unremitting attention and great patience.

In hospitals the dangers of admitting such cases to Wards for the treatment of non infectious diseases should always be carefully kept in view for there are many recorded instances of epidemics of diarrhoea arising in such Wards. In great Ormond Street Hospital for Sick Children a Ward of 14 beds is set apart for the treatment of this disease and the cases are nursed with every precaution for the prevention of the conveyance of infection by the secretions or excretions of the children; e.g. two or more nurses attend only to the removing of the soiled diapers; others attend only to the feeding; etc. Thus convalescent cases are safe guarded from reinfection and the nurses, themselves, are protected from the disease.

The value of fresh air for both patients and nurses in the treatment of this disease, cannot be too /

too strongly insisted upon. I always kept my ward windows open to their fullest extent and to this I attribute much of the immunity to skin infections, sore throats, etc which both patients and personnel enjoyed.

The treatment of acute infectious diarrhoea may be divided into stimulant and curative. The stimulant measures I mainly employed were hypodermic injections of strychnine, brandy, or ether; hot baths, sometimes, with the addition of mustard; and infusion with normal saline to which I usually added a few drops of a sterile solution of Adrenaline Chloride.

Hypodermic injections:- The hypodermic injection of strychnine is a very valuable method of combating collapse. I used the B.P. solution of Liquor Strychninae - 1% in doses of 1, 2, or even 3 minims, as an initial dose depending in part upon the age of the patient and in part upon the degree of the collapse. According to necessity I repeated it in 1 or 2 minim doses, 2, 3, or 4 hourly. It can be quickly, safely, and easily administered. There is no danger of poisoning so long /

long as the patient is carefully watched. I did not always stop the administration when twitching of the fingers appeared or convulsive movements of the limbs for these are symptoms which may arise independent of Strychnine in the course of this disease. They frequently occur in cases of collapse and I did not therefore consider them an indication for the withdrawal of the Strychnine but rather for the administration of more saline. However, under such circumstances strychnine must be given with great care.

Brandy and ether especially the latter, are not suitable for subcutaneous administration to infants. They exert a powerful local irritant action and I occasionally saw abscesses form at the seat of injection. They are more rapid and more transient in their action than Strychnine and were used mainly in cases of sudden dangerous collapse, often with excellent results, 5 10, or 15 minims were given as a dose. These drugs were not used in repeated injections. As a routine practise ten to twenty minims of brandy were added to the infant's feeds. /

feeds. It was supposed to act as a stimulant but I never saw any good or bad results from its use. Hot baths:- Hot baths are often invaluable. They however involve a certain degree of exposure and handling of the infant which in very severe cases may be dangerous.I had two cases where infants died while in hot baths so in cases of profound collapse I rely upon infusion and hypodermic injection of Strychnine, etc. and only give hot baths when the patient has rallied somewhat. Usually I prescribed them once a day, or more rarely night and morning. In cases with subnormal temperature they are very valuable. They may be given in one of two ways.

First, an infant having a rectal temperature of, say, 95°F is plunged into water at 101°F and kept there for 15 to 20 minutes. The rectal temperature is found to rise attaining progressively 96, 97. 98, and 98·4°F.

Or, second. the infant is plunged into water which has a temperature one degree higher than that of its body, that is 96°F. The heat of the water is gradually increased till it reaches 99°F; the infant's temperature is at the same time steadily rising, reaching /

reaching ultimately 98·4°F.

It is found that the temperature of an infant remains much longer near the normal and falls much more slowly when it is kept for a time in water made progressively warmer up to a certain point than when it is kept throughout in water of the same high temperature.

Sometimes, the temperature of the bath is raised to 105° or even 110°F and the child seems to derive benefit from the resulting cutaneous stimulation. Mustard was usually added to these baths to induce a powerful cutaneous vascular dilatation and thus aid in the redistribution of the blood which had accumulated in the dilated splanchnic vessels.

Not only in cases of subnormal temperature are hot baths useful but also in cases of pyrexia. I have often noted in cases of high temperature-, 103°, 104°; 105°F, a fall of 1°, 2°, or even 3°F after a hot bath.

## INFUSION.

### Normal Saline Adrenalin.

Chief among therapeutic measures to combat collapse is infusion. It is especially valuable in this disease for not only does it dilute the circulating toxines but it replaces the tissue fluids which have drained into the bowel, and which are essential to the continuance of life. Fluid is the greatest want of these little patients, and thirst the main cause of their suffering. Great improvement, therefore, is often seen in acute diarrhoea after infusion. My practice was to give four, five, six or eight ounces of normal saline subcutaneously, in cases in which, on admission the patient seemed collapsed. I, usually, infused in the mammary region. As the subcutaneous tissue there, is loose the fluid diffuses quickly and, without much pain, eight ounces can be infused. I never infused into the skin over the abdomen, for on two occasions I have seen difficulties of diagnosis arise owing to the muscular immobility and rigidity of the abdominal wall caused by the pain of such an infusion. I tried intravenous infusion several times, but it is attended with considerable difficulty; the superficial veins in an infant are small,

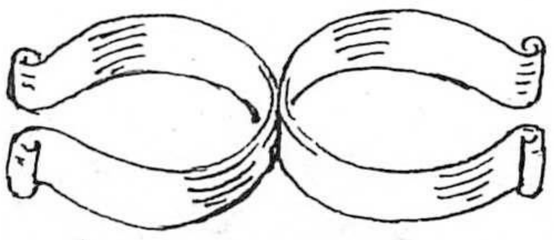
and in this disease owing to the small amount of fluid circulating in the vessels, the veins are collapsed. I usually infused into one of the deep veins of the upper arm. The beneficial effect was almost immediate and very gratifying. It is the method 'par excellence' but it requires considerable manipulative skill.

Whether intravenous or subcutaneous infusion be practised, the effect is transitory. The infusion must be repeated. Some index of the need of the body for fluid may be gained in the case of subcutaneous injection by observing the rapidity with which the saline is absorbed. If it is greedily soaked in by the tissues, then the infusion can safely be repeated after a few hours if necessary. If, on the contrary, absorption is slow, then a longer interval, one, two, or three days should be allowed to elapse. I had a case which I infused, at first, twice and later once daily. In six days I had given it 66 ounces of normal saline. I observed that from the third day the site of infusion was still oedematous 24 hours later. The child died suddenly on the sixth day. Its right heart was found to be greatly distended and its lungs were

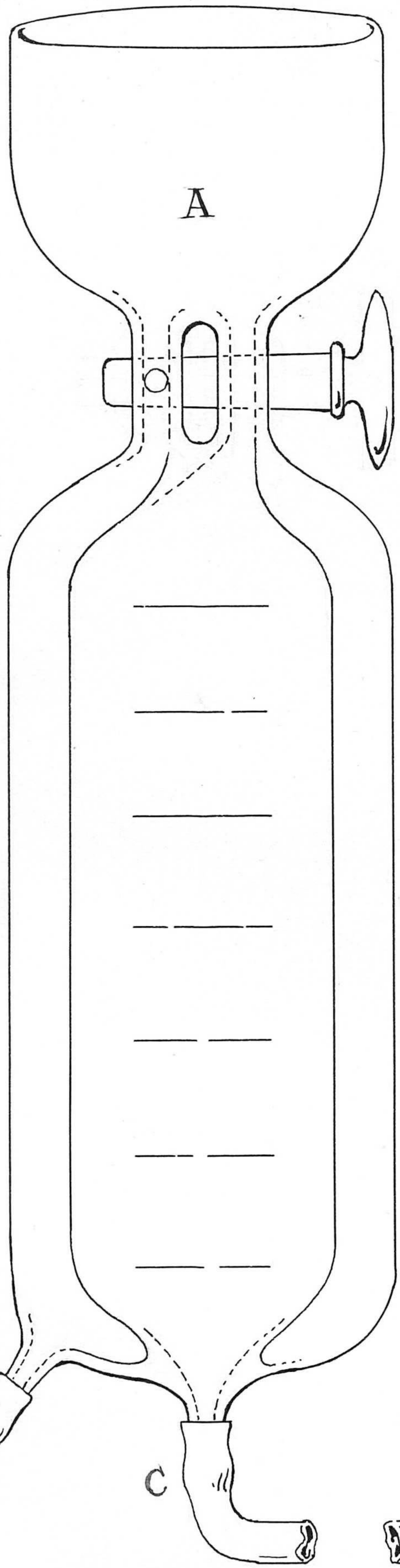
oedematous. I then made it a rule never to give more than 8 ounces in 24 hours and never to repeat an infusion until the last traces of the previous one had disappeared at least six hours.

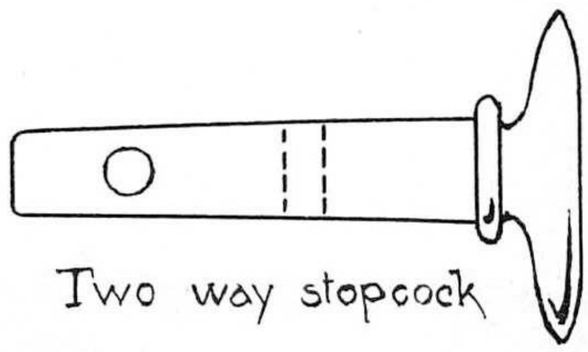
When I went to Great Ormond Street Sick Children's Hospital, the apparatus for infusing consisted of the glass barrel of a large syringe on the nozzle of which a piece of india rubber tubing was fixed so as to connect the barrel to a large sized hypodermic needle. The normal saline was poured into the barrel at a temperature of 140°F; it flowed through the tube and on issuing from the needle it was found to be 99°-102°. As the same tube and needle were always used it was thought that the fluid always entered the infant's tissues at this temperature, 99°. But the rate of infusion varies greatly in infants for the looseness of their subcutaneous tissues and the avidity with which the saline is absorbed are not constant. Moreover it took from 15 to 45 minutes or even longer for eight ounces of normal saline to pass into an infant's tissue through a needle of the calibre we used. I found it a practical impossibility to maintain the fluid in the syringe barrel at 140°F. throughout this time, and the saline often entered the tissues

# Saline Adrenalin Infuser

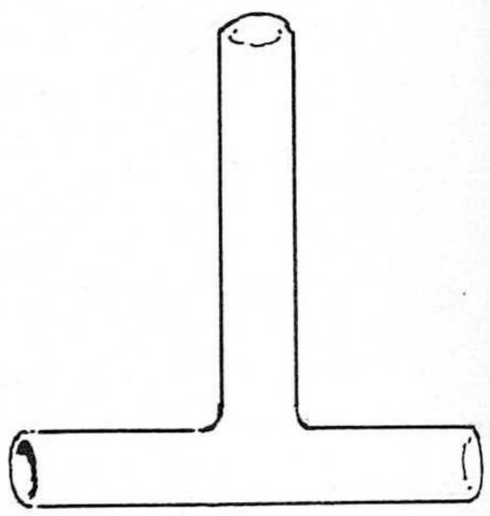


Clip (reduced)

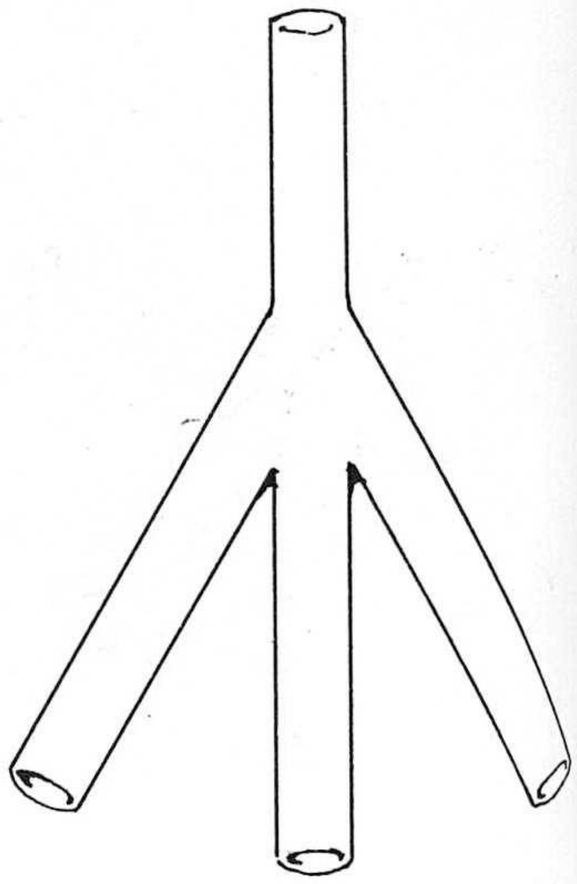


Two way stopcock



T perce.



3 way tube.

PLATE V

at a temperature much lower than that of the body. To this fact I attributed the occasional failure of infusion to induce a rally. There were other drawbacks to the apparatus we used; we could never accurately tell how much saline had been given for we were continually pouring out and renewing the contents of the syringe so as to ensure the proper temperature of the saline; again, dust from the air had free access to the uncovered fluid and although we exercised every precaution in the preliminary cleansing of the site, etc., yet occasionally abscesses formed there. To obviate these difficulties, and to simplify the procedure, I devised a little instrument which was made for me by Messrs. Baird and Tatlock, Edinburgh. The following is a short description of it:- It consists of a small receiver A joined by a small neck to a graduated container B. The container has double sides which divide it into an inner and an outer compartment; the inner contains the fluid to be infused, the outer the fluid which will keep the temperature constant. The inner container has an egress tube C. to which the india rubber tubing carrying the needle is attached (if the symptoms are very urgent and speedy infusion is desired then it

can be carried on simultaneously at two or three sites by attaching the glass T piece or 3 way tube to C and then connecting them with two or three hypodermic needles); the outer container has an egress tube D to which a piece of india rubber with a clip is attached so that the contents of the outer container may be run off at will, when they become cooled. A two way stop cock connects the receiver with the inner or outer container as desired.

It is used thus:- the apparatus is sterilised by boiling; the stopcock is turned to connect the receiver A. with the outer container which is then filled with water at, say 140°F. The stop-cock is then turned so as to connect the receiver A with the inner container and the latter is then filled with the infusing fluid which should be at such a temperature that the saline issuing from the needle is not lower than 98° nor higher than 101°. The inner container is made of a capacity of 8 ozs; it should be filled to begin with and at the end of the infusion the amount given can readily be calculated by subtracting the amount remaining in the container from 8 ozs. The fluid in the outer container is renewed as often as may be necessary to maintain the infusing fluid at the desired temperature. If the apparatus be wrapped in wool

it maintains its heat longer. The connections should be as short as possible so as to obviate cooling. The apparatus can be fixed to any crib by means of a double 'u' shaped clip, one arm of the 'u' grasping the apparatus while the other encircles one of the upright bars of the crib.

The apparatus was made of glass specially annealed to stand boiling which added considerably to its cost. I have not yet had an opportunity of using it very extensively. It seems, however, admirably adapted to fulfil the three purposes for which I devised it:-

1. To maintain the temperature of the infusing fluid at the necessary height.

2. To enable the amount infused to be accurately estimated.

3. To obviate contamination of the saline by dust, etc.

If it continues to serve I shall popularise an unbreakable form in tin or other metal so as to ensure cheapness and durability.

Professor John Chiene and others have laid stress upon the value of adrenalin as an adjuvant to

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 29 | Gladys Martinelli | 5/12 | Goldsmith | |

Disease

Result Death.

| Date | | September 10 | September 11 |
|---|---|---|---|
| C° | F° | M: E | M: E |
| | 107° | | |
| | 106 | 3 | 4 |
| 41° | 105 | Vomits. | |
| | 104 | Mustard Bath. | Saline Adrenalin Infusion 3vi |
| 40 | 103 | | |
| | 102 | | |
| 39 | 101 | | |
| | 100 | | |
| 38 | 99 | | |
| | 98 | | |
| 37 | 97 | | |
| | 96 | | |
| 36 | 95 | | |
| 35 | | | |
| Pulse | | 120 | 142 |
| Resp. | | 36 | 28 |
| Bowels | | 1 | 4 |
| Weight | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| September 10 | Alb. Water 3i hrly. Brandy mx hrly. | Stomach and rectal lavage daily. 5 pm. Strych. inj. hyp. (1 in 400). mj if 2 hrly for 8 doses. | | | |
| September 11 | | 4 am. Saline Adrenalin infusion 3vi. Strych. inj. hyp. (1 in 400) mj iij. | | | |

normal saline in infusing for the shock following operation, haemorrhage, etc. I have used it extensively in the collapse of acute diarrhoea, I consider that many of my cases derived considerable benefit from it and some rallied in a most wonderful manner. Its effects are not permanent; they seem to pass off within 4, 6, 8, or 12 hours depending in part upon the dose given and in part upon the initial condition of the patient. I have never used it in this disease apart from normal saline so it is impossible for me to pronounce upon the degree of benefit which the drug produced. Only once did it fail to prolong life more than twenty four hours after admission.

Case 27. Gladys Martinelli, 5/52; diarrhoea and persistent vomiting for five days. Bottle fed from birth - cow's milk and barley water; bowels moved 2, 3, 4 and 5 times per day habitually; a brother is a patient here for chronic diarrhoea.

Physical Examination; Collapsed; occasional rhonchus over bases; neck and legs stiff; drowsy; knee jerks active; no eye signs. Lumbar puncture negative.

Temperature febrile; died within 24 hours.

Treatment - routine and adrenalin (See Chart 29 )

Post mortem - negative.

The saline alone is such a powerful stimulant that too much credit must not be given to the Adrenalin but the combination seemed to me to act quicker and more powerfully. As it will tend to produce a general vascular constriction, it will relieve the dilated and congested condition of the splanchnic vessels, promote a general vascular equilibrium, increase the blood supply to the central nervous system and thus counteract shock. I think it is a very valuable adjuvant to the treatment of infantile diarrhoea. At first I used it in doses of 1-3 minims of a solution whose strength was 1 in 10,000. I increased the dose to 10 minims without observing any but beneficial results and then I used a solution of 1 in 1000 in doses of 1-2 minims. The adrenalin was added to the eight ounces of normal saline so that a dose of 2 minims of a 1 in 1000 solution was given as eight ounces of a 1 in 1,920,000 solution.

I never saw any ill effects from it. I never considered myself justified in delaying infusion to experiment with adrenalin alone in cases of grave collapse. The cases in which I think it did good are described in other parts of this thesis under otherheadings. The amounts given

are noted on the charts, and I have recorded the saline adrenalin infusion, in red, alongside the temperature. I would particularly draw attention to those charts where the opsonic index is marked. It will be at once seen how often a rise, sometimes of considerable extent, followed immediately on a saline adrenaline injection; as for its alleged effect in causing haemorrhage not only has it absolutely no pharmocological basis but, I think, it has not even a basis in fact. Haemorrhage was noted in two of my cases:

Case 28 , Maud Lismore: 5/12; intermittent diarrhoea since birth; present attack of vomiting and acute diarrhoea began yesterday. Premature - 7 months. Cow's milk and boiled water - peptonised - since aet. 5 weeks. Three other children - all dead.

1st - dead born.

2nd - died aet. 5 weeks of "wasting".

3rd - died aet. 10 months, bronchitis.

She was very collapsed on admission; the pulse was imperceptible at the wrist; there was slight ptosis on the right side ; slight strabismus; the eyes were sunken, the features pinched, the fontanelle very depressed; the heart feeble and rapid; and the extremities cold. The motions were frequent, green

and very foetid; there was occasional vomiting; the temperature was 97°; the abdomen was tumid with resistant walls; neither spleen nor liver could be felt; and there were a few moist crepitations at both bases.

A saline adrenalin infusion was given immediately on admission. After a few hours a subcutaneous haemorrhage was noticed at the site of infusion and it gradually spread till it occupied an area of about 4 square inches.

The patient rallied markedly after the infusion. The temperature remained normal throughout the disease. Vomiting however became so persistent that nasal feeding was resorted to, but without avail. Death took place 7 days after admission.

The cause of this haemorrhage was obviously accidental. I gave all infusions myself and carefully chose a site which was free from superficial veins. This was the only case of haemorrhage at the site of infusion, although I gave a great number of saline adrenaline infusions. This accident has been frequently observed in infusions with ordinary saline, and in this case I think its cause is patent. I had punctured a superficial vein with the needle. Post mortem only the usual changes in acute diarrhoea were found.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 30 | Ronald Sired | 3/12 | Goldsmith. | |

Disease

Result Death.

| Date (September) | 24 | 25 | 26 | 27 |
|---|---|---|---|---|
| Pulse | 128 | 128 | 120 | 124 |
| Resp. | 36 | 48 | 40 | 38 |
| Bowels | 2 | 3 / 2 | 1 / 1 | 3 |
| Weight | | | 6lbs 12. | |

Saline Adrenalin 3vi. Vomit. Adrenalin mi (1 in 1000) 4 Centimeters (Liquid)

0 0 0 3 not (blood)

| Date | Diet | Treatment |
|---|---|---|
| Sep. 24th | Alb. Water 3j<br>Brandy mxx hrly.<br>Nutrient Enemata every 4 hours.<br>Omit Brandy | Sep. 24.<br>Stomach and rectal lavage.<br>Strych. inj. hyp. (1 in 100) mij 2 hrly for 3 doses.<br>Infusion Saline Adrenalin 4/j 3 vj. |
| Sep. 25th | Alb. Water 3i 2hrly. | Sep. 25.<br>Resp. Strych. for 3 doses.<br>12 p.m. Resp. for 4 doses |
| Sep. 26 | a.m.<br>Alb. Water 3ii 2 hrly<br>p.m.<br>Alb. Water 3j hrly. | Sep. 27.<br>Resp. Strych for 4 doses. |

In the other case the bleeding was also due to adventitious circumstances.

Case 29. , Ronald Sired, 7/12; diarrhoea and vomiting for 14 days; premature - 7 months - weight at birth 5½ lbs. Breast fed for first 8 weeks ; then cow's milk and barley water.

First child died - "consumptive bowels".
Second child - dead born.
This - the third child.

On admission he presented all the typical symptoms of extreme collapse; he had no eye signs; the abdomen was tumid and resistant but neither spleen nor liver were enlarged; the nasal bones were thickened; there were no mucous patches and no rash. He was immediately given a saline adrenalin infusion (see Chart 30 ). He was vomiting violently, and after the infusion had been given brown specks were noticed in the vomit which on examination I found to be blood. The stomach was washed out after the blood had been noted in the vomit. The patient died suddenly 4 days after admission. On post mortem examination the mucous membrane of the stomach was found to be slightly congested; that of the oesophagus was much more congested and its surface was rough . There was

no congestion in the rest of the alimentary canal. Neither liver nor spleen was enlarged and both seemed normal. There was no evidence of congenital syphilis found.

The time between the administration of the saline adrenalin infusion and the appearance of the haematemesis was 20 mins. The infusion had then been only slightly absorbed and but an infinitesimal amount of adrenalin was then circulating in the blood. By what mechanism did it produce the haemorrhage? Did it also cause the congestion of the oesophagus? Why is this the only instance? How did the other cases in which I gave it escape? Only prejudice could conceive any causal connection between the haematemesis and the administration of adrenalin.Acute congestion and bleeding from the alimentary tract is not very rare in infantile diarrhoea. I have cited several instances (page ) among my own cases where no adrenalin had been given and I consider haematemesis would have occurred in this case whether adrenaline had been administered or not.

These were the only cases in which any untoward symptoms were observed where adrenalin was administered. Its use was always followed by immediate

if temporary benefit. I consider its sphere is to quicken and enhance the effect of the saline infusion in tiding over the condition of collapse in acute infantile diarrhoea and thus allowing time for the exhibition of more permanent remedies.

I tried continuous and intermittent rectal saline infusions in several cases but in too few to allow of any expression of opinion as regards the merits of the method compared with the subcutaneous.

## Curative Measures.

As soon as the patient has rallied somwhat from the collapsed condition, in which unfortunately most of our cases of summer diarrhoea are first presented to us, the stomach and rectum ought to be washed out. There is an acute intoxication present which arises from fermenting food remains from arrested digestion; altered secretions, and ptomaines, acids and other injurious products of bacterial action. Vomiting and diarrhoea are nature's methods for ridding the intestine of its contents and we should follow her guidance and thoroughly evacuate the stomach and bowels.

Stomach Lavage:- This is a very simple and efficacious procedure. A piece of india rubber tubing of somewhat large bore (No. 14), with holes at the sides of the end which is to be passed into the stomach is attached to an ordinary glass funnel of medium size. The tube, having been moistened is introduced as far as the base of the tongue. After the infant by instinctive movements has caused it /

it to enter the oesophagus it is gently pushed on till having traversed a course, including the mouth, of about 10 inches it reaches the stomach. Some people endeavour to wash out the stomach by means of a nasal tube if it is small enough to pass the nares of an infant a few months old it is too small for the passage of curds and mucus and is soon blocked.

I have rarely seen any harm result from the passage of the stomach tube. In two cases I saw inhibition of respiration but the child breathed normally again immediately the tube was withdrawn. The fluid used may be normal saline or a solution of bicarbonate of soda, two grains to the ounce. The washing out should be continued till the fluid returns clear. A few ounces of fluid should be left in the stomach and the tube quickly withdrawn. The effects of stomach lavage are often immediate and very gratifying. Vomiting usually ceases; the child's pulse becomes stronger and slower; the restlessness and anxiety are greatly calmed and the patient often falls into a deep sleep.

I was interested to note how little alteration there /

there was in the degree of abdominal distention in these cases after passage of the tube and washing out of the stomach and rectum. It seems to be due mainly to distention of the transverse colon.

Rectal Irrigation is also of great value in these cases. The moistened tube must be passed up nearly to splenic flexure. The pressure of fluid is usually about 12 inches. Irrigation is continued till the returning fluid is clear. I usually left about 3 or 4 ounces of fluid in the rectum. Boiled water or normal saline at a temperature of 100°F may be used and there is no limit to the quantity. I tried continuous rectal irrigation by means of a small apparatus. I devised but the results were not commensurate with the labour involved. In cases of prolonged diarrhoea with mucus in the stools the use of 3 or 4 ounces of a 0·5% solution of protargol after irrigation is beneficial.

Drugs in acute diarrhoea are not of much service. One by one they are gradually falling into disuse. In the early stages purgation is of great value and for this purpose few drugs excel Castor Oil /.

Oil. I used it largely in the out patient practice of the Hospital. In my experience it is quite exceptional to find it cause vomiting; it sweeps out the whole intestinal canal, it causes practically no griping; its action is certain and its after effects are soothing. I used it in doses of 2 to 4 drachms once or twice daily. Calomel in repeated small doses, 1/6 grain 4 hrly and grey Powder in doses of ½ grain twice daily are also of value. They are superior to the Salines for they not only purge but they also exert an antiseptic action on the intestinal tract. Salines such as Magnesium sulphate, Sodium sulphate or citrate, etc., may be used in small doses - ¼ - ½ drachm at intervals so that about one drachm is given three hourly. Tannogen and other astringents are of little or no value in acute infective diarrhoea. Opium is also valueless and, at the same time, an extremely dangerous drug to give to infants especially those suffering from collapse. The gravity of the condition is due to the toxaemic not to the diarrhoea. As I have already remarked diarrhoea may /

may be altogether absent. Treatment which aims only at diminishing the diarrhoea is therefore futile, irrational, and harmful.

Intestinal Antiseptics are of little service. In doses sufficient to exercise any marked effect on intestinal fermentation they produce local irritant and general toxic effects. Their value has been much over-estimated. If one considers the area of intestine and the bulk of contents upon which they are expected to exert an influence it will be obvious that they can have little action in the doses in which they are usually administered. Those which are soluble affect only the stomach and upper part of the small intestine; those which are insoluble, Salol. Resorcin etc., the lower part of the small intestine and the colon.
The best of them is probably salol but I never saw much good effect even from its use. I gave it to children under one year in 1 - 2 grain doses three times per day. Many clinicians land their favourite remedies for this disease Bismuth, Acids, Alkalies, etc.. but their descriptions are more convincing of /

of their therapeutic prejudice than of any scientific basis for their faith. We made an extensive trial of a new method of treatment: it consists in the feeding of cases of acute infective diarrhoea upon milk which has undergone prolonged lactic fermentation and to which I have given the name of "lactated" milk. I shall now proceed fully to detail and discuss this treatment.

## "LACTATED" MILK.

From time immemorial the beneficial effect upon intestinal conditions of milk which had undergone lactic acid fermentation has been universally recognised. In Ireland, Wales and some parts of Scotland under the form of "buttermilk" it is a valued factor in domestic therapeutics; in Germany as "diche Milch" it is a much lauded food in dyspeptic conditions; as Koumiss, the product of an alcoholic and lactic acid fermentation of mare's milk, prepared by the Arabs, it is used in the irritable digestive states produced by phthisis and other debilitating diseases, and among many nations, in analogous conditions the value of similar preparations - "matzoon" in America , "lelaen" in Egypt, "kefir" in the Caucuses - has long been recognised. Of late years the question of the value of lactic acid and lactic acid forming organisms has been the subject of elaborate investigations in the Paris Medical School. Professor Metchinkoff showed that most lactic acid forming organisms were non pathogenic to man and that some forms had the power of inhibiting the

growth and action of certain pathogenic and putrefactive organisms. Hayem, Lannelongue, Widal and others have used it extensively, in many gastric and intestinal diseases with considerable success. It has been shown that lactic acid bacilli, when given by mouth, develop in the small intestine producing their characteristic fermentation, reducing the putrefactive intestinal processes, as shown by the diminution in indican and ethereal sulphates in the urine, and conferring a distinct acid reaction on the stools.

Chemical substances as intestinal antiseptics in the diarrhoea of infants had proved practically worthless for in the doses requisite to exercise a salutary effect on intestinal fermentation they produced local irritative and general toxic symptoms. A powerful yet innocuous intestinal antiseptic, such as lactic acid bacilli which moreover in virtue of the acidity of their product might also be expected to modify the biliary and pancreatic secretions, seemed to promise no uncertain measure of success. My visiting physician, Dr. Batten, decided to give it an extensive trial and the following are the details of the experiment:-

We used a fluid preparation called "lactobacilline", a commercial product prepared by a

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Parisian firm "Le Ferment". It is a broth culture of two organisms (1) a long rod-like bacillus growing in chains; staining with gram, and known as B. Acidi lactici, Bulgarian variety, (2) a short rod form known as Streptococcus acidi lactici, European variety. Fresh sterilised milk at a temperature of 96° F. was inoculated with the lactobacilline - one tubeful - about 3 fluid drachms to each eleven ounces of milk. It is mixed well, incubated at 96° for seven hours, and then placed in an ice chest for 12 hours. The result is a creamy fluid of an agreeable acid state, to which I gave the name of "Lactated milk". The chemistry of this product is not definitely known.

According to M. Fouard of the Pasteur Institute, analyst for the proprietors, each litre of "lactated" milk contains 10-16 grams of pure lactic acid and about 50% of the caseine and 80% of the calcum phosphate present in the fresh milk is rendered soluble. In using it I found the most suitable strength for the initial dose was six drachms of lactated milk to one ounce of water; then I gave equal parts of water and lactated milk and continued to reduce the proportion of water till the patient was getting pure lactated milk.

It was used in twenty one cases, some of which for various reasons, did not constitute a fair test; in four it was successful but only one of these presented the typical picture of acute infantile diarrhoea; six improved temporarily; and three became markedly worse. Recovery therefore occurred in 19 per cent; one of these was a case which was not acutely ill and which I consider would have improved under ordinary routine treatment; this reduces the percentages of cures to 14.2 which does not markedly differ from my percentage of recoveries, 12.8 under routine methods of treatment. These figures are not encouraging but although these results do not justify my faith, I feel convinced that with further experience this combination of a bacteriological agent and an easily assimilable food will prove a powerful remedy in diarrhoeal conditions in children.

Infants showed a great tolerance for "lactated" milk in spite of the fact that the customary dose of two ounces two hourly was equivalent to about 64.4 minims of B.P.Acidum Lacticum (75%). The motions improved markedly; in some cases green stools became yellow in a single night; a greenish

tinge however reappeared in them on exposure, but whether this was the result of light, oxidation, or fermentative change, I was not able satisfactorily to determine. In children who had been under its influence for more than ten days, the stools became slimy. Three cases developed stomatitis, in one of definite membranous character. Two of them died and the affection was found to have spread to the oesophagus and stomach. The bacteriological examination negatived diphtheria and it is probable the condition was due to the corrosive action of the acid on tissues whose resistance was weakened by the existence of an acute infective condition. In two cases severe vomiting and collapse followed the administration of lactated milk. I shall now give in detail the cases thus treated.

A. Cured -

Case 6, : Gladys Pinchbeck, 1 1/12 years, ill for 10 days, diarrhoea for nine; vomiting occasionally. Breastfed plus bread and milk; "wasting" and cough for several months.

There had been severe pain on passing a motion and on one occasion 9 days ago, the stools had

| No. 10 | Name Gladys Punchbeck | Age 1 1/12 | Ward | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Cure. |

| Date (September) | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | October 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Temp. M. | 3 | 4 | 2 | 2 | 0 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Temp. E. | 2 | | | | | | | | | | | | | | | | | | | |
| Pulse | 144 | 140 | 132 | 132 | 116 | 120 | 120 | 130 | 120 | 128 | 124 | 140 | 132 | 148 | 156 | 130 | 116 | 120 | 122 | 128 |
| Resp. | 28 | 24 | 24 | 32 | 28 | 24 | 28 | 20 | 24 | 28 | 28 | 40 | 28 | 28 | 32 | 36 | 40 | 48 | 40 | 40 |
| Bowels | 3/3 | 3/2 | 3/1 | 1/2 | 3/1 | 2/1 | 3/1 | 1/2 | 0/0 | 1/1 | 1/1 | 0/1 | 1/2 | 1/0 | 0/1 | 2/2 | 1/2 | 1/2 | 1/2 | 2/1 |
| Weight | st: lbs oz 1.. 0. 8 | | | | | st: lbs oz 1.. 0. 8. | | | | st: lbs. oz. 1.. 0.. 8. | | st: lbs. oz. 10.. 12 | | | | st: lbs. oz. 1.. 1.. 8. | | | | |

Vomits.

Protargol - 0.5% - rectal lavage 4 oz. bis die

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| Sept. 15 | milk ʒ ii<br>Water ʒ i<br>Brandy mv XX.<br>2 hrly. | Sept. 15 Stomach and rectal lavage<br>mist. Bismuthi Co ʒi<br>4 hrly. | | | |
| Sept. 16. | Albumen Water ʒiii<br>2 hrly. | Sept. 18<br>Rectal lavage - Protargol<br>4 oz of 0.5% solution bis die. | | | |
| Sept. 18 | Lactated milk ʒii<br>Boiled Water ʒi<br>2 hrly. | Sept. 20<br>Lotio Hydr. Perchlor. fort. ears.<br>mist. Pot. Chlor. as mouth wash | | | |
| Sept. 23 | Lactated milk ʒii<br>2 hrly. | Sept. 21<br>Omit rectal lavage. | | | |
| Sept. 24 | Milk ʒiii ʒi<br>Lime Water ʒiii mi<br>Cream mi X<br>2 hrly. | Glycerophosphate ʒi in ʒi<br>as mouth wash.<br>Gradually increase strength<br>to ʒiii in ʒi water. | | | |
| Sept. 25 | milk ʒiii 1½<br>L. W. ʒiii i<br>Cream mv XX<br>2 hrly. | | | | |
| Sept. 26 | milk ʒiii ij<br>L. W. ʒiii i<br>Cream mv XX<br>2 hrly. | | | | |
| Sept. 27 | milk ʒiii 1½<br>L. W. ʒiii i<br>Cream mv X<br>2½ hrly. | | | | |

contained several ounces of bright red blood. Latterly, the motions had been streaked with blood.

On admission the child seemed markedly ill; the abdomen was not distended; vomiting and diarrhoea were frequent and the motions were brown, liquid, slimy, very offensive, and streaked with blood. Rectal examination proved negative. The child was first placed for 24 hours on two ounces of milk and one of boiled water 2 hourly. As this was not well tolerated, three ounces of albumen water were given instead. Then, 72 hours after admission the child was placed upon two ounces of "lactated" milk, and one ounce of water, two hourly. The motions rapidly improved and by the fourth day they were yellow, formed, inoffensive, and of normal appearance. On the seventh day, the lactated milk was stopped and ordinary milk and lime water substituted. The patient steadily gained weight. She remained in hospital five weeks and when discharged she had gained 1 lb. 12 ozs.

What share had "lactated milk" in this success? Very little stress can be laid on this case for, as is seen from the chart, three rectal injections each of 4 oz. of a 0.5 per cent solution

of protargol had been given just at the time when the patient began to improve (see Charts 10 & 11).

Case 7, : William White, 14/12; vomiting for ten days, diarrhoea for five; breast fed for 3 months; since, potatoes, broth, bread and milk, etc.; intermittent diarrhoea for many months.

On admission the patient seemed somewhat collapsed, but the pulse was well marked. The liver and spleen were enlarged; the chest rachitic; the muscles very flaccid; and there were only five teeth.

After stomach and rectal lavage and 24 hours of albumen water, lactated milk was given, at first 1 oz. diluted with 1 oz. of water, two hourly. The stools lost their green, slimy, offensive character, and became yellow and almost inodourless although still remaining fluid. There was no vomiting. After passing through various phases, the "lactated" milk was stopped on the 8th day. The child was discharged three weeks after admission in perfect health and with an increase in weight of 1½ lbs.

I have not thought it necessary to include

this chart. The result may or may not have been due to "lactated" milk. The disease was not typical of acute infective diarrhoea; it resembled more a chronic condition arising from prolonged bad feeding and the regular routine of the diet in hospital may have contributed most materially to the success of the case.

Case 8 : Annie Fryer; $^{20}/_{12}$; diarrhoea and vomiting for four days. Breast fed for three months; then Nestle's milk and nursery biscuits for one year; then family diet.

On admission the child was found to be considerably collapsed though not emaciated; the abdomen was tumid and soft; neither liver nor spleen were enlarged; the buttocks were not erythematous; and the patient had twelve teeth.

The collapse was sufficient to indicate saline infusion; the stomach and rectum were washed out; the bowel was rested for 24 hours with albumen water, and then "lactated" milk was given in 1 oz. doses two hourly. The green and fluid motions rapidly disappeared; the stools became of normal frequency - indeed, a certain amount of constipation developed,

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 12 | Annie Fryer | 1 8/12 | | |
| Disease | | | | Result Cure |

| Date (September) | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 140 | 128 | 136 | 120 | 100 | 104 | 96 | 100 | 164 | 96 | 100 |
| Resp. | 32 | 20 | 28 | 24 | 24 | 28 | 24 | 24 | 28 | 24 | 20 |
| Bowels | 1 | 3 / 3 | 3 / 2 | 0 / 2 | 0 / 0 | 0 / 0 | 0 / 1 | 2 / 1 | 1 / 2 | 2 / 1 | 2 |
| Weight | | | st. lb. oz. 1. 4. 8 | | | | | | | st. lb. oz. 1. 5. 8 | |

Vomit 3.
1
1
1
0
0
0
0
0
0
0
Vomit 3 vi.
Infusion 3 vi

| Date | Diet | Treatment |
|---|---|---|
| Sept. 7 | Aër. Water 3ii 2 hrly. | Sept. 7 Stomach and rectal lavage. Strych. inj. hypo. mjiii. for one dose. Infusion Saline 3vi |
| Sept. 8 | Lactated milk 3i Boiled Water 3i 2 hrly. | |
| Sept. 12 | Milk 3iii i Lime Water 3 i 3 hrly. | Sept. 12 ol. Ricini 3ii |
| Sept. 14 | Bread and butter | |
| Sept. 16 | Milk pudding and eggs. | |

| Date | Diet | Treatment |
|---|---|---|
| | | |

MARGIN TO BE LEFT FOR BINDING.

consistency, and colour. The patient was discharged cured in ten days having gained 1½ lbs. This is my only striking case of the complete success of "lactated" milk in acute infective diarrhoea (see Chart No. 12 ).

Case 9 : Edward Dell, 1 year: diarrhoea for a fortnight, vomiting for a day. The diarrhoea had been subacute, 3 to 4 motions per day, fluid and yellow, till the day of admission when, with the onset of vomiting it suddenly became very severe; he was a bottle fed baby, reared on cow's milk; for three months his diet had been general. His motions were green, frequent, slimy and offensive; vomiting was not a prominent symptom. After the routine treatment for 24 hours, he was placed upon lactated milk, 2 ozs. two hourly. His stools rapidly became normal and he was discharged in 8 days cured.

*

From these four cures, I do not think any reliable deductions can be drawn. In three, the motions were slimy, which signifies a certain degree

of chronicity in the diarrhoea. Only one of the four, case No. 8 , was considered sufficiently ill to require saline infusion; it was the only successful case of acute infantile diarrhoea thus treated and, therefore, is without significance.

From these results "lactated" milk seems to be beneficial in subacute diarrhoeal conditions, but it is open to question if it excels other remedies for this purpose.

B. Temporarily Improved:-

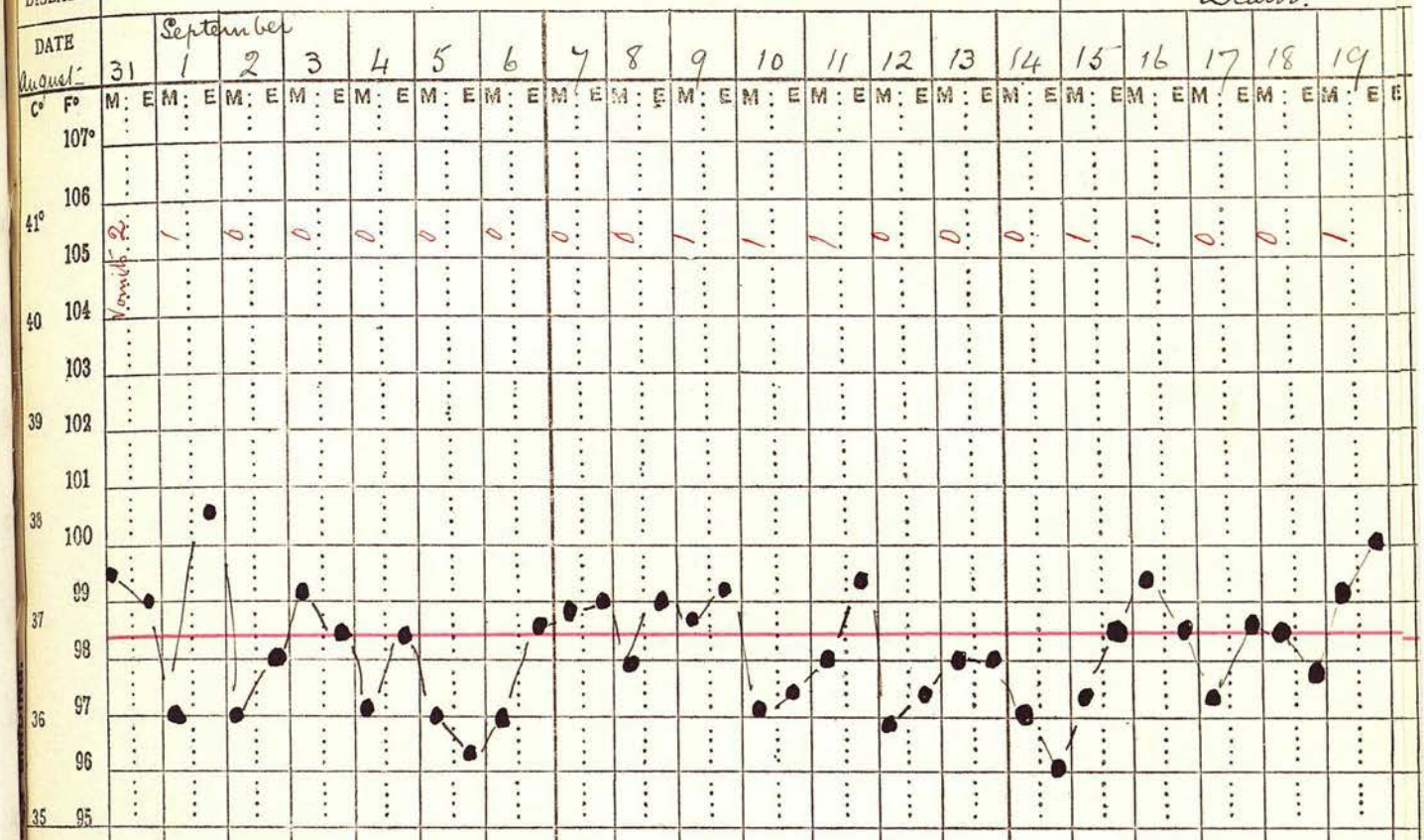
Case 10 : Edward Kelsey, 13/52; three weeks ago had diarrhoea for three days. After recovering from this attack he was put back on cow's milk when the diarrhoea immediately recommenced; there had been no vomiting. Bottle fed from birth - cow's milk.

Family history:- 1st child died - diarrhoea and vomiting.
2nd child died - broncho pneumonia.
3rd child alive - rachitic
4th child is the patient.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 13 | Edward Kelsey | 13/52 | Goldsmith | |

Disease

Result Death.




| | Aug 31 | Sept 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 104 | 96 | 100 | 96 | 100 | 100 | 96 | 88 | 96 | 100 | 104 | 120 | 110 | 124 | 116 | 108 | 88 | 104 | 128 | 120 |
| Resp. | 24 | 24 | 28 | 24 | 24 | 24 | 28 | 24 | 20 | 24 | 20 | 52 | 40 | 62 | 44 | 40 | 40 | 36 | 40 | 50 |
| Bowels | 0 | 1 / 1 | 2 / 2 | 3 / 3 | 2 / 3 | 2 / 2 | 4 / 2 | 2 / 3 | 2 / 3 | 2 / 2 | 2 / 3 | 3 / 1 | 1 / 1 | 1 / 1 | 2 / 2 | 2 / 1 | 3 / 2 | 4 / 3 | 2 / 3 | 3 / 4 |
| Weight | | 9lbs 12 | | | 10lbs 4 | | | | | | | 10lbs 4 | | | | 10lbs 12. | | | | |

| Date | Diet | Treatment |
|---|---|---|
| Aug. 31 | Alb. Water 3ii 2 hrly. | Aug. 31 Daily Stomach and rectal lavage. |
| Sept. 1 | Lactated Milk 3vi<br>Boiled Water 3i 2 hrly. | Sept. 3. Mist. Pot. Brom. 3ii rep. p.r.n. |
| Sept. 4 | Lactated Milk 3iX<br>Boiled Water 3vii 2 hrly. | Sept. 19 Str. Inj hyp. 1 in 100 mii H hrly. |
| Sept. 12 | Lactated Milk 3iv<br>Boiled Water 3i | Sept 20. Rep. mii 3 hrly. |
| Sept. 14 | Lactated Milk 2 hrly. 3ii to 2 hrly. | Sept. 21. Omit Str. Mist. Pot. Brom 3ii |
| Sept. 17 | Lactated Milk 3ii<br>Boiled Water 3iv | Sept 22. Mist. Pot. Brom 3i 1 hrly. |
| Sept. 22 | Brandy 2 hrly. mXXI in each feed. | Sept 26. Hot baths + massage bis die. |
| Sept 23 | Lactated Milk 3i<br>Boiled Water 3i 2 hrly. | Sept 27. Omit rectal lavage. Alkaline lotion for nose. |
| Sept. 24 | Peptonised Milk (30 min) 3i<br>Lime Water 3i 2 hrly. | Sept. 29 Mist. Sp. Aeth. ℥ Ammon 3i tid |

| Date | Diet | Treatment |
|---|---|---|
| Sept. 26 | Pepton. Milk 3ii<br>Lime Water 3i 2 hrly. | Oct. 5 Inj hyp. Str. ch. 1 in 400 mii 2 hrly. |
| Sept. 29 | Pepton. Milk 3ii<br>Lime Water 3i<br>Brandy mXX 2 hrly. | Mustard Bath<br>Ether mX R.p.m. |
| Oct. 2 | Whey 3ii 2 hrly. | |
| Oct. 5 | Veal Tea 3½ in each feed. | |

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 14 | Edward Kelsey | 13/52 | Goldsmith. | |
| Disease | | | | Result Death. |

| Date, September | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | October. 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| M | 1 | 1 | 1 | 1 | 1 | 2 | 0 | 0 | 0 | 0 | 1 | 0 | 4 | 0 | 0 | 0 |
| E | 1 (vomit) | | | | | | | | | | | | | | | |
| Pulse | 100 | 100 | 128 | 112 | 140 | 108 | 120 | 120 | 108 | 116 | 120 | 136 | 120 | 134 | 140 | 146 |
| Resp. | 40 | 40 | 48 | 40 | 44 | 32 | 40 | 56 | 48 | 36 | 40 | 40 | 36 | 36 | 44 | 40 |
| Bowels | 3/4 | 3/1 | 2/3 | 2/3 | 2/3 | 2/1 | 2/1 | 2/2 | 2/2 | 2/4 | 2/4 | 2/0 | 2/5 | 2/3 | 2/2 | 2/ |
| Weight | 10lb 4 | | | | 10lb | | | 10lb 4 | | | | 9lb 12. | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

On admission the motions were found to be green, slimy, and offensive; they contained Morgan's bacillus. The child weighed 10 lbs. 4 oz.; the buttocks were erythematous and the spleen and liver were enlarged.

After the usual preliminary treatment the patient was placed on "lactated" milk (For details see Chart 13 ). At first he throve; the motions became normal and he gained 1 lb. in weight. But after a time a change appeared; the motions while still remaining yellow became slimy and he seemed to go rapidly downhill. He reached his maximum improvement on September 16th, and between that date and September 24th, he lost 1 lb . in weight. I thought the long continued use of a highly acid diet had possibly deranged his metabolic processes, so I abruptly changed him on to a strongly alkaline food - peptonised milk and lime water. He rallied for about eleven days. Then a profuse nasal discharge appeared which on bacteriological examination was found to contain "many short and long bacilli resembling Klebs-Loeffler. But few cocci were seen in the films; the cultures were swamped by yeast cells."

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 15 | Philip Bennett | 5/12 | Goldsmith. | |

Disease

Result Death.

7.

| Date, September | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| M. | | | | | | | | | | | | | | | | | | | | |
| E. | 1 | 1 | 0 | 1 | 2 | 0 | 3 | 3 | 3 | 1 | 0 | 1 | 2 | 1 | 1 | 1 | 4 | 1 | 0 | 0 |
| Pulse | 120 | 124 | 88 | 100 | 96 | 100 | 124 | 100 | 124 | 128 | 124 | 120 | 124 | 122 | 120 | 124 | 128 | 124 | 116 | 124 |
| Resp. | 24 | 28 | 20 | 24 | 24 | 24 | 28 | 28 | 28 | 24 | 28 | 24 | 28 | 24 | 28 | 28 | 24 | 32 | 28 | 28 |
| Bowels | 1 | 3 | 2 | 2 | 3 | 3 | 3 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 3 | 2 | 1 | 1 | 2 | 2 |
| Weight | | | 9lb 8 | | | | 10lb | | | | | 10lb | | | | 9lb 8 | | | | 9lb 12 |

Vomit

| Date | Diet | Treatment |
|---|---|---|
| Sept. 8 | Whey 3ii 2 hrly. | Sept. 8 Stomach and rectal lavage daily. |
| | Brandy mxv. | |
| Sept. 12 | Lactated milk 3i | Sept. 11. Mist. Ipecac. c Ammon 3½ t.d. |
| | Water 3i 2 hrly. | |
| Sept. 23 | Lactated milk 3iii | Sept. 12. Omit Stomach lavage. |
| | Water 3i 2 hrly. Feed nasally. | Sept. 14 Resume Stomach lavage daily. |
| Sept. 24 | Milk 3i | Sept. 18 Hydrar. c Cret. grs ½ for one dose. |
| | Water 3i 2 hrly. | |
| Sept. 25 | Milk 3i | Sept. 23 Mist. Pot. Brom 3½ to be given before each feed if vomiting continue. |
| | Lime Water 3i | |
| | Cream mx 2 hrly | |
| Sept. 26 | Milk 3i | |
| | Lime Water 3i | |
| | Cream mxx 2 hrly | |
| | Peptonised 20 min. | |

| Date | Diet | Treatment |
|---|---|---|
| | | |

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 16 | Philip Bennett | 5/12 | Goldsmith. | |

Disease

Result Death.

| Date, September | 28 | 29 | 30 | October 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 112 | 116 | 120 | 132 | 124 | 116 | 112 | 128 | 140 | 136 | 152 | 144 |
| Resp. | 24 | 26 | 24 | 24 | 28 | 20 | 24 | 32 | 48 | 44 | 40 | 40 |
| Bowels | 2/2 | 3/1 | 2/4 | 1/2 | 2/5 | 2/1 | 1/1 | 2/2 | 1/2 | 0/5 | 3/4 | 4 |
| Weight | | | | 9lb 8 | | 8lb 12 | | | | | 8lb 12. | |

| Date | Diet | Treatment |
|---|---|---|
| Sept. 28th a.m. | Milk 3½<br>Lime Water 3j<br>Brandy mjXX<br>2 hrly. | Sept. 29th<br>Massage: Hot Baths bis die.<br>Stomach Lavage bis die. |
| p.m. | Peptonise for 30 min.<br>Albumen Water 3ij<br>Brandy mjXX<br>2 hrly. | Oct. 2nd<br>Mustard Bath p.r.n.<br>Inj. Strych. hyp (1 in 400) mjj 2 hrly for 4 doses |
| Oct. 4th | Lactated Milk 3j<br>Water 3j<br>2 hrly. | Rep. for 4 doses.<br>Ether mjX p.r.n.<br>Oct. 3rd Rep. Strych. for 4 doses.<br>Oct. 4th " " " " "<br>Oct. 5 " " " " "<br>Oct. 6 " " " " "<br>Oct. 7 " " " " "<br>Oct. 8 " " " " "<br>Oct. 9 " " " " " |

| Date | Diet | Treatment |
|---|---|---|
| Sept. 6th | Peptonised milk and Benger's Food 3ij 2 hrly nasally.<br>Brandy mjXX | |
| Oct. 8th | Omit Benger's.<br>Cream mjX in each feed. | |

The patient sank rapidly; the motions continued yellow, fluid, and inoffensive; a few moist crepitations appeared at both bases and the patient died on October 6th. There were no symptoms of diphtheritic paralysis. Unfortunately, I could not obtain a post mortem examination.

This was a most disappointing case. "Lactated" milk cured the local intestinal condition and yet the patient died. Had I produced a profound derangement of the normal metabolic processes by the long continued use of an excessively acid diet? Did the child succumb to the effects of the toxaemia which is always present in acute infective diarrhoea and which so often produces in cases which survive the primary acute attack, a well marked marasmic state? Or was death due to diphtheria or some allied disease?

Case 11 : Philip Bennett, 5/12; diarrhoea and vomiting for six weeks. Breast fed till six weeks ago - Nestle's milk, cornflour, arrowroot, etc. since.

On admission patient was in a somewhat collapsed

and emaciated condition, the result of the long continued subacute diarrhoea. The weight was only 9 lbs. 8 ozs; the buttocks were erythematous; the liver and spleen were enlarged and the motions were frequent, fluid, green, and offensive; they were said to be sometimes cheesy, sometimes slimy. The patient was vomiting after every feed; he was put upon "lactated" milk (see Charts 15 & 16.) and his stools became yellow and inoffensive and he gained weight but gradually he,like the last case, began to fail; vomiting became pronounced on September 24th, so lactated milk was immediately stopped, after 12 days administration. He then rallied for a time but gradually sank again and died a fortnight later.

The post mortem showed no pronounced pathological changes except great thinning of the intestinal wall.

Why did this child die? Had it also been left too long on "lactated" milk?

Case 12 : Florence Davies, $^{14}/_{52}$; diarrhoea for 3 days, vomiting for 2. Bottle fed from birth - Nestle's milk, Quite well until present attack.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 17 | Florence Davies | 2/12 | | |

DISEASE

Result Death.

| Date August | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | September 1 | 2 | 3 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | | 152 | 140 | 120 | 132 | 128 | 144 | 120 | 132 | 120 | 96 | 140 | 120 | 116 | 140 | 140 | 140 | 112 | 104 | 120 |
| Resp. | | 52 | 40 | 40 | 48 | 48 | 44 | 40 | 32 | 40 | 36 | 52 | 52 | 32 | 40 | 40 | 56 | 24 | 36 | 40 |
| Bowels | 1 | 3 / 2 | 4 / 4 | 3 / 2 | 2 / 2 | 4 / 3 | 3 / 2 | 3 / 2 | 2 / 3 | 3 / 4 | 4 / 4 | 4 / 3 | 5 / 3 | 3 | 2 / 3 | 3 / 3 | 3 / 3 | 3 / 3 | 3 / 3 | 3 / 3 |
| Weight | | | 9·8 | | | 9·8 | | | | | | 9·4 | | | | | | 9·8 | | |

Ophthalmic Index.

Horse Serum 5 c.c.

| Date | Diet | Treatment |
|---|---|---|
| Aug. 15 | alb. Water ℥ i<br>Brandy ♏ v hrly. | Aug. 15 Daily stomach and rectal lavage<br>Saline Infusion ℥ vi<br>Liq. Hyp. Strych ♏ ii hrly for 6 doses. |
| Aug. 17 | alb. Water ℥ i<br>Whey ℥ i<br>Brandy ♏ v hrly | Normal Horse Serum Subcutaneously 5 c.c. bis die |
| Aug. 19 | Whey ℥ ii 2 hrly | Aug. 20. Rectal injection Protargol 0·5% ℥ iv daily. Omit Stomach lavage. |
| Aug. 22 | Acid Cream ♏ x 2 hrly | Sept. 3. Protargol per os. Grs 1/10 4 hrly |
| Aug. 23 | Whey ℥ iii<br>Cream ♏ xx 2 hrly. | Sept. 5. Daily stomach lavage |
| Aug. 24 | Peptonised Milk ℥ ii 2 hrly. | Sept. 18. Mist: Pot. Chlor. ℥ i 4 hrly. |
| Aug. 27 | Milk ℥ i<br>Lime Water ℥ i<br>Cream ♏ x 2 hrly. | Sept. 19. Omit Mist. Pot. Chlor. Alkaline lotion for throat<br>Mist. Ipecac. & Ammon ℥ ½ 4 hrly. |

| Date | Diet | Treatment |
|---|---|---|
| Aug. 29 | Citrated Milk ℥ i<br>Lime Water ℥ i<br>Cream ♏ x 2 hrly. | |
| Aug. 31 | Whey ℥ iii<br>Cream ♏ v 2 hrly. | |
| Sept. 1 | Whey ℥ ii 2 hrly | |
| Sept. 3 | Alb. Water ℥ ii 2 hrly. | |
| Sept. 4 | Whey ℥ ii nasally 2 hrly | |
| Sept. 7 | Omit nasal feeds. | |
| Sept. 9 | Lactated Milk ℥ i<br>Boiled Water ℥ i | |
| Sept. 20 | Lactated Milk ℥ ½ 2 hrly<br>Boiled Water ℥ ½<br>Brandy ♏ xx 3 hrly. | |

REMOVED TO BE LEFT FOR BINDING.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 18 | Florence Davies | 2/12 | | |

DISEASE

Result Death

| Date September | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 152 | 144 | 140 | 132 | 112 | 136 | 128 | 124 | 140 | 144 | 130 | 156 | 128 | 140 | 130 | 140 | |
| Resp. | 36 | 36 | 48 | 40 | 32 | 40 | 36 | 40 | 32 | 44 | 32 | 38 | 36 | 36 | 28 | | |
| Bowels | 2/3 | 2/4 | 3/3 | 4/3 | 3/4 | 3/5 | 2/3 | 3/2 | 2/2 | 2/3 | 3/2 | 2/2 | 2/2 | 3/1 | 2/3 | 4/4 | 4 |
| Weight | | | | 9.2. | | 9.4 | | | | 9.8. | | | | 9.8 | | | |

Hyoscine Tinden.

Hyoscine 0.01

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

WARNING. TEMPERATURE TO BE TAKEN FOR SIGNING.

On admission she was so collapsed as to require saline infusion; her motions were frequent, fluid, foetid, but yellow and contained Morgan's bacillus; she was vomiting after every feed. First she was treated on Albumen water, whey, peptonised milk etc. On September 8th, she was given lactated milk for her motions were still offensive and frequent (7 per day) and she was occasionally vomiting. A marked improvement immediately took place in the character and frequency of the stools and the vomiting practically ceased (see Charts 17 & 18). But two days later September 10th, I first noticed a white membrane on her soft palate and uvula. Again the bacteriological report was not definite as to whether the infection was diphtheritic or not (see also Case 13 ). The membrane spread to the tongue, fauces, and pharynx, but the submaxillary and cervical glands were not enlarged. The child steadily sank; symptoms of laryngeal obstruction appeared which led me to intubate. The child died on September 20th; the following is the result of the post mortem examination:-

Brain:- slight congestion - no oedema nor meningitis.

Ears:- Mucopus present in both ears; membrane congested but not perforated on either side.

Mouth:- There was merely a white deposit on the tongue and tonsils - no membrane.

Oesophagus:- Throughout its entire length were numerous small white areas the size of a split pea, covered with a white film which could be scraped off leaving a white surface. Films and culture from this deposit showed a short rod like bacillus resembling the Klebs-Loeffler bacillus.

Larynx:- showed a slight swelling of the epiglottis but no trace of membrane.

Heart:- wall of left ventricle slightly thinned; otherwise normal.

Lungs:- slight oedema and congestion in both bases.

Stomach:- slight congestion of the mucosa; no evidence of patches such as were met with in the oesophagus, excepting over a small area near the cardiac orifice.

Intestine:- no congestion; walls thin; Peyer's patches swollen but not injected; no ulceration; follicles in upper part of the large intestine somewhat enlarged.

This was a most instructive case. Again, there

was a decided improvement for a time under "lactated" milk and then the child seemed to waste away in spite of all our efforts to feed it. There are several examples of this secondary fatal condition among my other cases of diarrhoea and I have seen analogous cases in "Les Enfants Malades" at Paris. This striking occurrence in four of the twenty one cases of "lactated" milk treatment, affords a valuable suggestion for any future trials of this remedy- viz. that in all cases in which "lactated" milk is administered to infants, its use should cease as soon as it has made the stools yellow and inoffensive. Prolonged exposure to "lactated" milk causes inanition. This is probably due not to faulty assimilation for the stools of such infants remain of normal colour and consistency on other foods such as peptonised milk. I had not time, nor opportunity to prove this point by chemical analyses of the excreta. At the Clinique Tarnier I had considerable experience in analysing stools in cases of defective assimilation, and from the experience I there gained of the appearance of such stools I am strongly of opinion that the characters of the motions in these three cases showed that assimilation was not at fault. The question is

somewhat complicated by the occurrence of a low form of membranous inflammation in two of the cases, No. 12 and No. 13 . Its precise nature was never satisfactorily determined. Cultures of the "lacto bacilline" yielded a profusion of varieties of organisms and I think it very probable, under the especially favourable conditions for bacterial growth, presented by the greatly lowered vitality of the tissues of an infant which has just survived an acute attack of infective diarrhoea, that some normally, non pathogenic forms of these lactic acid bacilli developed sufficient virulence to give rise to the diphtheroid appearances observed in the mucous membrane. In other words, I think that the membranous condition was without determining effect upon the issue of the disease and that these cases from a less obvious cause were doomed. No membrane was detected on post mortem examination in Case and yet it ran a precisely similar course. The mechanical effect of the membrane in obstructing the entrance to the air passages undoubtedly hastened the end, but it is not unlikely that a fatal termination was in any case, inevitable. It is interesting to note the peculiar distribution of the membrane in the stomach in Case No. 12 . It occurred only

at the cardiac end where there are no acid forming glands. Was the organism capable of flourishing in a concentrated solution of lactic acid and yet unable to survive exposure to an attenuated solution of hydrochloric acid? The causal organism of this condition may have had an origin extraneous to the "lactated" milk, but it is not probable seeing the membranous condition appeared only in these two cases, during three months in a ward of fourteen beds, devoted exclusively to the treatment of "summer diarrhoea". As all the milk was first sterilised it could not have been the source of the infection . We must, I think, incriminate the "Lacto bacilline" and in my future use of this or any such substance my first precaution will be to ensure the purity of the lactic acid strain or strains of which it is composed.

If we eliminate faulty assimilation, the toxaemia arising from the diarrhœa and the toxic effect of the organisms which give rise to these membranes, there only remains as the cause of the fatal inanition a defective metabolism due to the ingestion of an excessive amount of acid. I previously pointed out that the daily dose of lactic acid which many of these infants received was equivalent to about 64.4 minims of B.P. Acidum Lacticum (page 89 ).

The strength of B.P. Acidium Lacticum is 7.5 per cent so this is roughly equally to 48 minims of pure lactic acid, $C_3H_6O_3$.

Lactic Acid is monovalent -

$$NaOH + C_3H_6O_3 = NaC_3H_5O_3 + H_2O$$
$$40 \qquad 90 \qquad 112 + 18.$$

40 grams of sodium hydrate would be equivalent to 90 grams of lactic acid. So the daily dose of 48 minims of lactic acid which these infants ingested would be equivalent to the alkalivity of 13.8 c.c. of a deci normal solution of sodium hydrate. I cannot say how much of this acid was absorbed and I have been unable to find any reliable data as to the alkaline potentiality of the blood of a normal infant.

Three other cases improved temporarily under "lactated" milk. They were not, however, uncomplicated cases of summer diarrhoea, so I shall not discuss them here.

Case 13 : Gladys Brandon, $^{12}/_{12}$; intermittent diarrhoea and collapse of 4 days duration; no vomiting. Breast fed till a fortnight ago; cow's and Nestle's milks since. On admission patient was emaciated and collapsed; the motions were fluid, frequent, offensive, and of a brownish green colour; mouth coated with "thrush"; broncho-pneumonia at left base; spleen palpable; abdomen tumid; urine negative; heart regular but feeble; knee jerks absent. This case was treated on lactated milk for 8 days, receiving 1 oz. two hourly. The motions improved but after the first few days the patient steadily lost ground. There was a congested condition of the fauces present but no Klebs-Loeffler bacilli were detected in the throat swab. An ulcer appeared on the left side of the uvula and spread to the soft palate. Multiple stapylococcal abscesses developed on head, back, neck etc. Patient gradually wasted away and died seven weeks after admission. The post mortem examination threw

no additional light on the case.

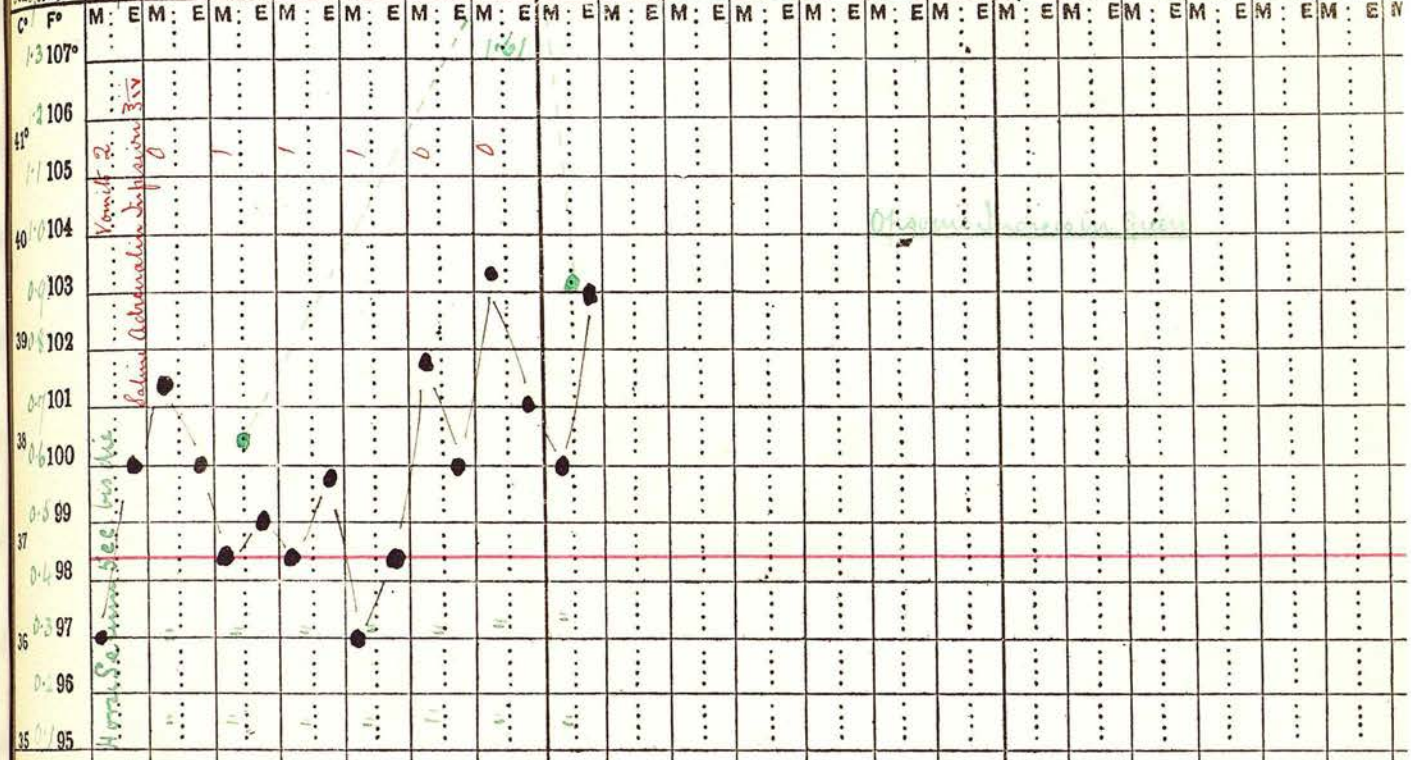
The improvement in this case, excepting in the condition of the stools, was very transitory. The lactated milk was given only in small doses and only for 6 days. Owing to the pulmonary condition and the multiple staphylococcal infection which were present together with the diarrhoea, I think there were too many complicating elements present to allow of any deductions being drawn as to the action of "lactated" milk so I shall not consider this case in my discussion.

Case 14 : Charles Blackburn, 1 year; This proved to be a case of Gaertner infection but in spite of the persistent high temperature and great constitutional disturbance the motions for a long time maintained a normal colour and consistency (For further details see Gaertner Infection, Case No.1 , p. 45 ).

Case 15 : Albert Quiver, 5/12; diarrhoea and vomiting for two days. Bottle fed from birth - peptonised milk. Youngest of ten children, of whom two have died from wasting. "Fits" since 3 weeks old.

| No. 19 | Name Albert Quiver | Age 5/12 | Ward | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| Date (August) | 29 | 30 | 31 | September 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|---|---|---|
| Pulse | 132 | 120 | 100 | 80 | 100 | 104 | 112 | 100 |
| Resp. | 48 | 40 | 32 | 24 | 32 | 28 | 36 | 32 |
| Bowels | 1 | 2 / 2 | 3 / 2 | 2 / 1 | 2 / 2 | 4 / 2 | 4 / 2 | 3 / 2 |
| Weight | | | | 9lb. | | | | |




| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| August 29 11 p.m. | Alb. Water 3j hrly. 3ij 2 hrly. Brandy ɱ X | Saline Adrenalin Infusion 3vi Stomach and Rectal lavage Inj. Strych. Hyp. 1 in 400 ɱij 2 hrly for 8 doses. daily. Serum 5 c.c. bis die. daily. Rep. Strych. | | | |
| Aug. 30 | Lactated Milk 3j Boiled Water 3j 2 hrly. | | | | |
| Aug 31 | Omit Brandy. | | | | |
| Sept. 1. | Alb. Water 3ij Brandy ɱ XV 2 hrly. | | | | |
| Sept. 2 | Lactated Milk 3j Boiled Water 3j 2 hrly. | | | | |
| Sept. 4. | | Omit Strych. | | | |
| Sept. 5 midnight | | Inj hyp. Strych - 1 in 400 - ɱij ondose. Saline Adrenalin Infusion 3vj. | | | |

On admission the motions were very foetid, frequent, and resembled water in colour and consistency; they contained Morgan's bacillus; vomiting occurred after every feed. There was a general convulsion just after admission and he remained rigid and cyanosed for several hours; the knee jerks were active, the plantar response extensor; lumbar puncture showed some leucocytosis; pulse slow; temperature febrile (see Chart 19 ) Purpuric rash and head retraction appeared on the 6th day and then death took place. (For treatment etc. see Chart No.19 .) In this case there was distinct improvement in the alimentary condition; the motions became yellow and inoffensive. The lactated milk was used only intermittently owing to difficulties of supply. Post mortem - a pneumococcal suppurative meningitis was found.

Post mortem:-

Brain - the vertex showed a thick, tough, yellow purulent exudate not entirely covering the gyri but disposed at the sides of the vessels as they ran in the sulci; at the base the purulent condition was most marked at the temporal poles where the brain substance was quite covered by exudate; only a small amount of pus present at the posterior part of the base. Down the sides of the

great longitudinal sulcus the vessels were surrounded by pus. The left half of the brain was opened and a small flake of pus was found on the choroid plexus. Cranial sinuses were normal.

Ears:- pus in both ears - the mastoid cells were filled; the membrane was not injected nor perforated. Cultures and films of pus from meninges and ears showed pneumococci.

Heart:- nil.

Lungs:- slight congestion at both bases.

Stomach:- small area of slight congestion - otherwise normal.

Intestine:- small gut, thin walled, empty and translucent mesenteric glands nil - large gut - follicles slightly enlarged.

From these three cases one fact stands out prominently as regards the action of "lactated" milk:- viz. in all, notwithstanding the severity of the general condition, the state of the alimentary processes improved markedly, under its use.

CLASS C. NO IMPROVEMENT.

Eight cases are comprised in this class. The

special feature in all of them was that "lactated" milk failed to tide them over the acute stage. Some, such as Case 61 [1] died within four days. One, Case 52 [2] was only on lactated milk for twenty four hours. Another, Case No.16 presented some interesting features (See Chart No.20 ):-

John Parker, 16/12; diarrhoea for 7 days; vomiting for two. Bottle fed from birth - cow's milk. On admission he was extremely collapsed; the motions were green, fluid, foetid and contained Morgan's bacillus; he was vomiting frequently thick, curdled, sour matter. He had 12 teeth; the liver and spleen were enlarged; there was a purulent discharge from the left eye; patient had a convergent squint; the pupils were circular the right was larger than the left; reaction unimpaired; convergent squint; right palpebral fissure larger than left; knee jerks active; no muscular rigidity; ears nil; lumbar puncture negative. Patient was placed upon routine treatment with the addition of "lactated" milk. The stools improved in character, and diminished in frequency, but the temperature continued febrile and patient died on the seventh day. There was a remarkable ante mortem

(1) See page ..... 223
(2) See page ..... 219

| No. 20 | Name John Parker | Age 1 4/12 | Ward Goldsmith | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| Date (Jan. Nov.) | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|---|
| Pulse | 100 | 120 | 124 | 120 | 130 | 100 | 128 |
| Resp. | 32 | 40 | 36 | 44 | 32 | 32 | 44 |
| Bowels | 2 | 2 / 2 | 2 / 1 | 1 / 1 | 0 / 1 | 1 / 3 | 3 |
| Weight | | | | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

rise of 10° F. within the last 36 hours. A post mortem examination was refused so the cause of this pyrexia remains obscure. Ear mischief so often a cause of high temperature in children, was here excluded; lumbar puncture proved negative; neither Widal nor Gaertner reaction was observed in the blood; so I have attributed the pyrexia to the intestinal condition and included this case in Class C. although it might suitably have occupied a separate class. The other cases in Class C. 38 , 43 , 52 , 61 ., 68 , are included in the Appendix.

CLASS D. MADE WORSE.

I have placed three cases in this category, 17 , 18 , and 19 . They all closely resembled each other, so I shall take No.17 as the type (see Chart No.21 ).

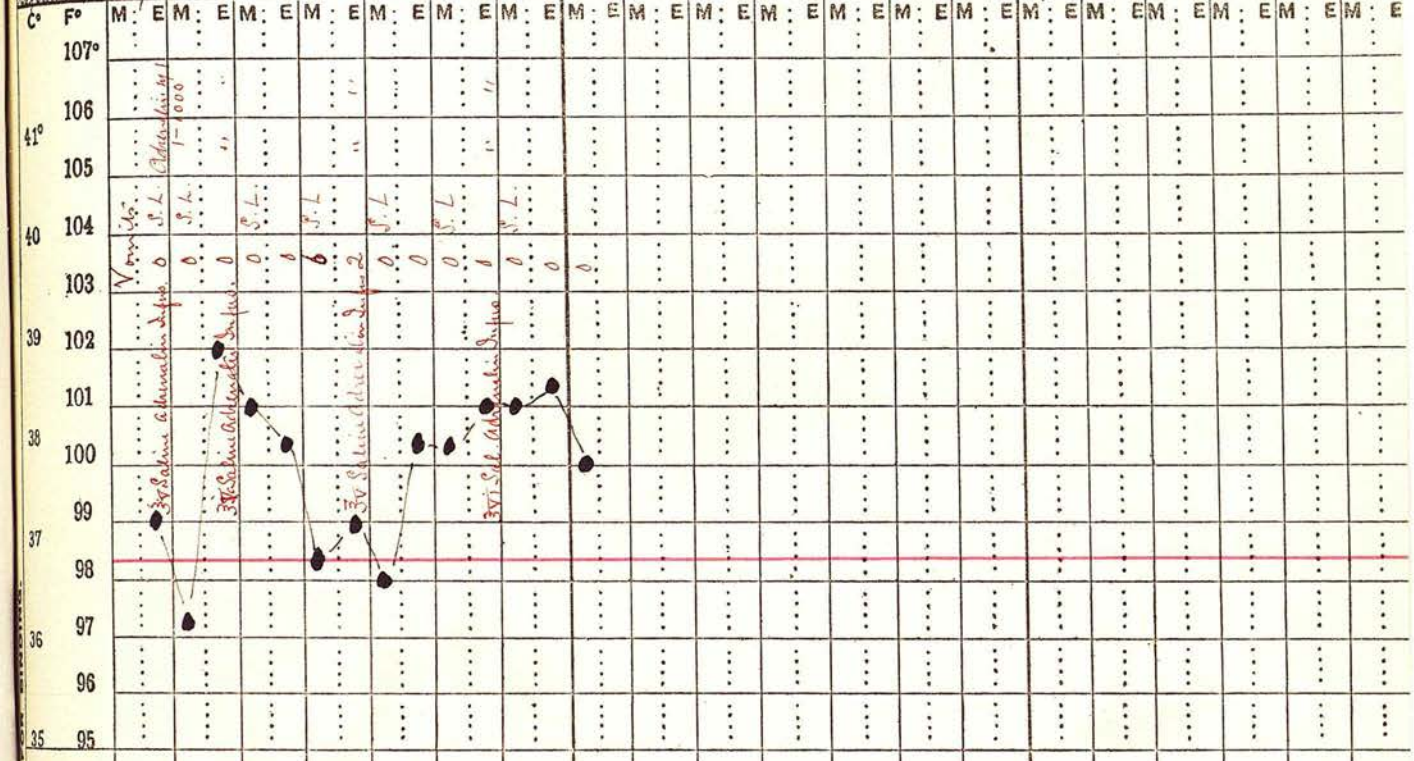
Otto Linck, 5/12; diarrhoea for 17 days, vomiting for 3. Breast fed for three months; then cow's milk and barley water equal parts, about eight ounces two hourly. On admission, this patient was extremely collapsed; the fontanelle was depressed; the eyes sunken; the pulse almost

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 21 | Otts Linck | 5/12 | Goldsmith | |

Disease

Result Death

Date (September / October): 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6




| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Pulse | | 132 | 120 | 144 | 124 | 116 | 120 | 120 |
| Resp. | | 40 | 44 | 52 | 40 | 36 | 32 | 44 |
| Bowels | 3 | 2 / 4 | 0 / 2 | 3 / 4 | 3 / 2 | 1 / 2 | 2 / 2 | 2 |
| Weight | | | | | | | | |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| Sept. 29 | Albumen Water 3i<br>Brandy mx hourly. | Sept. 29.<br>Stomach and rectal lavage.<br>Mustard Bath<br>Strych inj. hyp. 1 in 400 mxii<br>Saline Adrenalin Infusion 4 hrly. 3vii<br>Adren 1 in 1000 mxv. | | | Oct. 6<br>Omit Strych<br>Oxygen 5 min. hrly<br>Mist. Spirit. c. Ammon 3i t.i.d. |
| " 30 | Whey 3i<br>Alb. Water 3i<br>Brandy mxxx 2 hrly. | Later Mist. Pot. Brom 3i<br>3 doses. | | | |
| Oct. 2 | Whey 3ii<br>Brandy mxx<br>one feed only | Sept. 30.<br>1 am.<br>Saline Adrenalin Infus 3iv<br>Mustard bath<br>11-30 pm. Omit Strych. inj.<br>Inj. Ether mx<br>Mist. Pot. Brom p.m. 3i hrly. | | | |
| " 2 | Lactated milk 3i<br>Water 3i<br>2 hrly.<br>Later<br>Alb. Water 3ii<br>Brandy mxx 2 hrly. | Oct. 2.<br>Hyp. inj Strych 1-400. mij<br>Saline Adrenalin Infus. 2 hrly 3v<br>Mustard bath<br>Ether mx<br>1 dose. | | | |
| " 5 | Real tea 3iv per diem. | | | | |

imperceptible at the wrist and the extremities cold. The motions were green, foetid, and not very frequent, 4-5 per day. He rallied markedly after adrenalin saline injection; on albumen water the motions became yellow; then on the third day "lactated" milk was given; immediately he began to vomit violently and became greatly collapsed. "Lactated" milk was at once stopped and albumen water again given. He rallied again, but died suddenly three days later. On post mortem examination only the usual thinning of the gut with slight enlargement of the solitary follicles was found (See Chart 21 ).

Case 18 : Lilian Lindsay, 6/12; diarrhoea and vomiting for one month. Breast fed for three weeks, then Nestle's milk. The patient was in a very neglected state on admission, and somewhat collapsed. The motions were fluid brown and foetid, but not very frequent, about 4 per day. Vomiting was only slight; there were no teeth; the liver was enlarged; spleen not; the lungs were clear. After the usual routine preliminary treatment, the patient was put on "lactated" milk; the vomiting and diarrhoea became aggravated and the patient quickly sank and died within 36 hours. The temperature was markedly febrile throughout, varying from

97-103° F. No light was thrown upon the case by the post mortem examination.

Case 19 : Beatrice Stiff, 7/52; diarrhoea and vomiting for four weeks; ceased on August 20; recommenced August 26th. Breast fed for three weeks then bottle - cow's milk, 2 oz.,barley water 1 oz. two hourly. Weight 6 lbs. 14 oz. First child. On admission, August 29th, the motions were very frequent, like water and foetid; the patient was somewhat collapsed but improved markedly on adrenalin saline infusion. Physical examination was negative. Temperature was 99°. Albumen water was given for first twenty four hours, and then "lactated" milk 1 oz. boiled water 1 oz., two hourly was ordered. After the first dose the patient vomited violently, and in spite of strychnine and other stimulants, she did not recover from the resulting collapse, but died within 12 hours. The post mortem examination proved negative as also did the bacteriological examination of the stools.

In all three cases the fatal result seemed to be accelerated, if not caused by the administration of "lactated milk". In no case was it given undiluted, but, yet, it provoked acute intestinal

intolerance. The only wonder is that these cases were not more frequent, for we were giving a fluid which both from its concentrated acidity and from the fact that it contained fat and casein, seemed to be contra indicated in this disease. Hoping to ascertain the cause of this vomiting, I took the average daily amount of lactic acid, 64 minims, ingested by these infants; divided it into 8 doses, diluted it with an equal part of water, and administered it to two healthy infants. To one I gave it just before meals; to the other mid way between two meals. In both cases it produced vomiting. The vomited matters were sour and clotted. The second case had in addition, diarrhoea. I may not draw any very definite conclusions from this experiment for the results on the gastric irritability were complicated by the fact that the child was on a milk diet. The vomiting occurred sometimes immediately after administration, sometimes ten to fifteen minutes or even half an hour afterwards. But the vomited matter was always mainly curdled milk. I shall pursue this subject of infantile gastric tolerance to lactic acid and its reference to diet, at the earliest opportunity. I hope this summer also to determine in what degree the

bacterial action of "lactated " milk is responsible for the improvement so often noted in the stools and whether I could not get the same effect by combining lactic acid with predigested albumen, by filtering off any coagulum produced, and adding sugar and salts.

From this dietetic and therapeutic experimental study the following conclusions may be made:-

1. "Lactated" milk is usually well tolerated in acute infantile diarrhoea although exceptionally it may cause vomiting.

2. It has a decidedly beneficial effect upon the colour, consistency and frequency of the motions in this disease; it also diminishes vomiting.

3. It is a valuable remedy in more chronic forms of diarrhoea.

4. It should be continued only till the motions become normal in colour and reduced in frequency, for too prolonged use may cause an intractable and fatal form of marasmus.

5. Great care must be exercised in selecting the strains of lactic acid bacilli so as to obviate pathogenic action.

6. It is a remedy well worthy of further and extensive trial for with greater experience, in its application, it will probably prove invaluable in both acute and chronic diarrhoeal states of infancy.

Our main difficulty was to know how soon we ought to attempt to change from lactated milk to ordinary food. I am of opinion that the continuance of yellow stools for two days is an index that the intestinal flora has been modified sufficiently to warrant an attempt to reduce the amount of "lactated" milk. I tried many methods of effecting the transition to ordinary milk; reducing the amount of lactated milk; reducing the length of time during which it was incubated in preparation; increasing the dilution; diluting with whey, peptonised milk, sterilised milk etc. But I cannot say that any one method proved particularly advantageous . Different procedures seemed to suit different cases. And the only point I am certain about is that the change must not be abrupt. It ought to be begun as soon as the beneficial effects of "lactated" milk have been produced and it ought to proceed slowly and carefully.

As regards subsequent diet for children who have survived the acute attack of diarrhoea there are two essentials:-

It must have the maximum of nutritive power; and it must involve a minimum of digestive capacity. The intestine requires time and rest to recuperate; digestion and assimilation are almost non existent and yet the toxaemia and the fever have so depleted the tissues that if nutriment is not supplied in an assimilable form the patient will die from inanition. I made a series of experiments to find out what was the most digestible form in which I could administer cow's milk. In an acute attack it was customary to give what was called albumen water, the filtrate from the addition of white of egg to cold water. This contains a very small and variable amount of albumen and is chiefly of value as a source of fluid to the parched tissues. Clear whey was usually the next step in the diet ladder. It consists of lact-albumen, whey proteid, sugar and salts, and although possessing a certain nutritive value it is still far from adequate to support life. The next step usually is the most difficult, as it involves the addition to the diet of milk, and exposes the stomach to the strain of dealing with clots. I

endeavoured to find how best to treat milk so as to throw as little strain as possible upon the infant's digestive mechanism. I took a number of test tubes which each contained an equal bulk of milk and various diluents. To each I added an equal quantity of rennet and contrasted the physical appearances of the clots produced. Then I added various digestive ferments and incubated each tube with its control, at 37°C. for two hours. As all the tubes were then plunged into boiling water to destroy any further ferment action the results of the experiment could be permanently preserved without alteration by the addition of a few drops of formalin. The diluents used were, water, lime water, and a solution of sodium citrate. Lime water appreciably retarded clotting and gave a somewhat more flocculent clot. Sodium citrate was used in solutions of varying strength 1- 5 grains to the drachm and sufficient of this aqueous solution was added to the milk to give a strength of 1, 2 or 3 grains to the ounce. The presence of about 1 grain to the ounce of milk markedly retards coagulation: 5 grains to the ounce inhibits it. The coagulum was finer, more flocculent, softer and smoother /

smoother than that ordinarily produced by the action of rennet or milk. Infants readily tolerated larger quantities of milk if it were citrated. I found it very valuable in cases where the alimentary canal was still irritable after an acute attack of diarrhoea and in cases in which from prematurity or disease the digestive power was impaired. On citrated milk infants throve; they gained in strength and weight but I was very chary of the two prolonged use of an alkaline solution for I feared it might permanently affect the gastric secretion. Concerning the action of the digestive ferments I need not enter into details. Their results have been long well known. I used Park Davies & Co's preparations, Pepsin in a strength of $\frac{1}{4}$ gr to $\frac{1}{2}$ oz. and Pancreatin $\frac{1}{2}$ gr. to $\frac{1}{2}$ oz. The only point I would like to emphasise is the excellence and the completeness of the digestion of milk by Pancreatin in the above strength when the milk has been diluted with half its bulk of lime water; no further clot can be obtained by the addition of more Rennet and there is no precipitate on boiling. The fat and proteid have both been digested. Doubtless some of the product has little or no nutritive value but it contains a considerable proportion of the nutritive matter in a non irritant and perfectly assimilable form and is a most useful

inter /

intermediary step between whey and milk. When it had to be used as a food I predigested it for not longer than 30 minutes and as the child progressed I decreased the period of digestion in five minute stages till the infant was receiving undigested milk. The child was dismissed from hospital when it was progressing on a milk predigested for 10 minutes. The further stages were completed while it was under observation as an out patient. Too much reliance must not be placed on the nature of the stools as an index to the success of the feeding. They are certainly a valuable but not an infallible guide. The index of the success of the treatment is the condition of the patient and not of his stools and if the patient is gaining in strength and weight the diet should not be altered. Great care and patience is needed; too speedy an advance on the diet ladder may cause a relapse, while persistence on a low scale of nutrition may cause fatal inanition.

## NORMAL HORSE SERUM.

### Opsonins.

As my results from the use of this substance in cases of acute infantile diarrhoea were neither satisfactory nor conclusive, I shall only briefly refer to this part of my treatment. In this disease the patient's strength is so quickly exhausted that death frequently ensues before curative measures have had an opportunity to exert their influence. I have described various measures for stimulating these patients (see pages 62, 67, 74, etc.) so as to tide over the threatening collapse till curative treatment can be brought into operation. The place of horse serum among remedies in this disease was, I thought, midway between the temporising, symptomatic agents, and the curative. In it I thought I had substance which when injected subcutaneously, would supply a certain amount of readily utilisable nutritive material. But besides being a food it, I thought, might have a definite action in raising the resistance of the patient to the infection of this disease just as Professor Metchnikoff has shown it does in other conditions which led him to term it a "Stimuline" of the lympatic system.

I injected 5 c.c. of Burroughs & Welcome's normal Horse Serum twice daily. The injections were usually made into the skin in the mammary region. Where practical, the opsonic index of the patient was taken before injection and at regular intervals afterwards. The opsonic index of two cases, Violet Burrell, No. 20 and John Parker No. 16 , was observed throughout the course of their disease so as to obtain a standard in this affection in absence of horse serum. I shall exclude all cases which were not under this treatment for at least 48 hours. I had six of longer duration which I shall now briefly detail.

Case No.20 , Violet Burrell, $^{6}/_{52}$; habitually constipated; 14 days diarrhoea; vomited twice. Breast fed for first fortnight; Nestle's milk and barley water since.

Motions - green from commencement, 8-10 per day; contained Morgan's Bacillus; no vomiting.

Physical examination - negative, except slight enlargement of liver. Rallied a little at first; then became more and more restless; cyanosed; slight convulsive twitchings;, few moist sounds developed in base of left lung; no eye symptoms;

| No. 22 | Name Violet Burrell | Age 6/52 | Ward Goldsmith | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| Date | August 31 | September 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 124 | 136 | 136 | 128 | 126 | 120 | 136 | 120 | 140 | 136 | 100 | 132 | 108 | 116 | 120 |
| Resp. | 28 | 32 | 28 | 28 | 26 | 24 | 28 | 28 | 24 | 28 | 28 | 24 | 28 | 28 | 36 |
| Bowels | 1 | 0 / 1 | 1 / 2 | 2 / 2 | 2 / 3 | 2 / 2 | 4 / 3 | 4 / 2 | 4 / 3 | 2 / 3 | 2 / 4 | 4 / 5 | 3 / 4 | 3 / 3 | 2 |
| Weight | | 1 lb. 8 | | | | 8 lb. | | | | | 7 lb. 4. | | | | |

Vomits 3. 1 2 0 0 0 4 2 1 1 1 1 2 1 4

Saline Infusion 3VII — Saline Adrenalin Infusion 3VII — Saline Adrenalin Infusion 3V — Saline Adrenalin Infusion 3V — Saline Adrenalin Infusion 3V — Saline Adrenalin Infusion 3V — Saline Adrenalin Infusion 3V

| Date | Diet | Treatment |
|---|---|---|
| Aug. 31 | Albumen Water 3 ij 2 hrly. | Aug. 31. Stomach and rectal lavage daily. |
| Sep. 1 | Whey 3 j<br>Alb! Water 3 j<br>Brandy mx 2 hrly. | Sep. 3rd Strych. Inj. hyp. (1 in 400) mj ij 2 hrly. 12 doses.<br>Inf. Saline. 3 VIj |
| Sep. 3 | Alb. Water 3 ij 2 hrly. | Sep. 7th Rep. Strych. 2 hrly. |
| Sep. 5 | Lactated Milk 3 vj<br>Boiled Water 3 j 2 hrly. | Sep. 8th " " 4 hrly.<br>Infus. Saline Adrenalin. (mj-litre 1000) 3 VJ |
| Sep. 7th | Whey 3 ij 2 hrly. | Sept. 11th Mist: Ether Co Aqueous 3ij t.i.d.<br>Infus. Saline Adrenalin. 3 IV. |
| Sep. 8th | Lactated Milk 3 j<br>Boiled Water 3 j<br>Brandy mx xx 2 hrly. | Sep. 13. Infus Saline Adrenalin 3 IV |
| | | Sep 14. Oxygen p.r.n.<br>p.m. Oxygen min. 5. hrly. |

fists clenched; legs drawn up; gradually sank; died 15 days after admission; pneumonia at right base.

Weight on admission 7 lbs. 8; at death 7 lbs.4.

Post mortem:-

Lungs:- Some congestion and collapse at both bases patch of broncho-pneumonia, consolidation on right side involving back of lower lobe about its apex. Right pleura slighly adherent.

Liver and spleen:- normal.

Stomach:- somewhat distended.

Intestines:- No congestion - no swelling of Peyer's patches. Follicles in large intestine somewhat enlarged. Walls pale and thin.

Mesenteric glands - large and soft - not tubercular.

Brain - normal.

I had six observations of her opsonic index and I have charted them in green (See Chart No.22 ).

The Opsonic Index was remarkable for its extreme lowness; for four days it remained between 0.10 and 0.16. Then it began steadily to rise and continued to do so until death. It did not vary with the temperature. The ante mortem rise was probably due to the development of broncho-pneumonia which was the only marked lesion present, post mortem.

Case 16 , John Parker: This case has already been detailed (see page 109 ). Unfortunately, I have only two observations, one for September 5th 0.14 and the other for September 6th 0.44. Death occurred on September 7th (See Chart No.20 ). This observation again shows a very low reading with, so far as we can judge from two observations, a tendency to an ante mortem rise. This case showed an inexplicable rise of temperature to 107°F.

The standard is very imperfect but both cases show a pronounced tendency to low readings.

Of the cases I treated with horse serum, I shall first detail:-

Case 21 , Ethel Castle, 3/12; diarrhoea and vomiting for 3 days. Bottle fed from birth with cow's milk and barley water. On admission the patient was very collapsed; the motions were green offensive and very fluid but vomiting was not pronounced. Under treatment she imrpoved rapidly and was discharged cured after 17 days, on August 16th. The Opsonic Index had been taken on two occasions, August 13th and 15th, and was found to be 0.88 on both.

On August 21st, she was re-admitted almost morbund, she was vomiting incessantly sour, clotted

| No. 23 | Name Ethel Castle | Age 3/12 | Ward Goldsmith | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| DATE August | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 |
|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 120 | 140 | 136 | 160 | 160 | 144 | 180 | 160 | 140 | 150 |
| Resp. | 33 | 32 | 40 | 80 | 48 | 40 | 80 | 48 | 32 | 44 |
| Bowels | 1/4 | 0/3 | 4/2 | 5/3 | 3/4 | 4/2 | 3/5 | 3/3 | 3/4 | 4/ |
| Weight | | | | | | | | | | |

Opsonic Index

Horse Serum 5cc.

| DATE | DIET | TREATMENT | DATE | DIET | TREATMENT |
|---|---|---|---|---|---|
| | | | | | |

milk and had a profuse almost colourless diarrhoea. She was very restless but nothing could be detected on physical examination. The vomiting was very persistent in spite of repeated stomach lavage, and blood was several times noted in the product. The treatment was routine plus normal horse serum. The first striking point was that the patient who had been discharged with an Index of 0.88 returned after 4 days with an index of 0.54. A series of positive and negative phases are observed on the Chart (No. 23 ) which correspond more or less accurately to the temperature fluctuations, a high temperature, usually corresponding to a low opsonic index. So far as can be seen in the Chart there was an ante mortem rise and the general tendency of the Chart is downwards.

On post mortem examination, haemorrhages were found in the stomach wall and there were areas of acute congestion in the small intestine. There were somewhat enlarged follicles in the large gut. In the lungs, there was a patch of broncho-pneumonic consolidation at the left base which I had not detected during life.

Case No. 12 , Florence Davies (See page 98 for case details). There was no normal taken. The case was under treatment with horse serum for 29

days. The opsonic index was first observed 48 hours after the administration of horse serum had been begun (See Charts 17 & 18, page 98 ). It was 0.40 on August 17th, two days later a negative phase ensued, the O.I. falling to 0.10; this persisted for 24 hours and then it almost steadily rose, attaining a maximum of 0.76 on August 27th; then it declined and on September 5th the lowest figure 0.07 was noted; on September 8, there was a remarkable rise to 1.06 from 0.15; this was maintained for two days and then the index rapidly fell to 0.16; then it fluctuated for 24 hours; a well marked ante mortem rise took place of 0.55.

The fluctuations of the opsonic index in this case are very difficult to explain. They bear no relation to the temperature as can be at once seen on looking at the Charts 17 & 18.

Why the low readings occurred on September 19, 20, and 21, I do not know for the vomiting and diarrhoea had considerably improved, and the temperature was approaching normal. Horse Serum had been given since September 15th. The child continued to improve and a well marked positive phase ensued and lasted till September 27th. On that day

I changed the diet abruptly. The alimentary condition then gave me some anxiety, and the negative phase which occurred was possibly the consequence of this retrogression. I had to retrace all my steps in this infant's diet till on September 4th, I had even to institute nasal feeding on account of vomiting. On September 8th, I gave lactated milk for the first time and this corresponds to the great rise in the oponic index observed on that date. A gentle fall is noted over the next two days. On September 10th, I first observed a white membrane on the fauces (see previous notes page 99. ) From that day the patient rapidly sank, and the opsonic curve fell almost vertically to 0.16. The patient died on September 15th. The ante mortem rise is probably an expression of the last flicker of resistance of the dying organism.

Case 32 , Frank Taberner, 3/12; diarrhoea for 3 days, vomiting for 2; Breast fed for two months; then cow's milk and barley water equal parts - 8 tablespoonfuls two hourly. On admission he was very collapsed; his motions were copious, green, slimy, and foetid; about 8 or 9 per day, and contained Morgan's bacillus; he was vomiting after every

| No. 24 | Name Francis Tabernor | Age 3/12 | Ward Goldsmith | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |

| Date August | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 |
|---|---|---|---|---|---|---|---|---|
| Pulse | 140 | 144 | 128 | 100 | 152 | 143 | 160 | 140 |
| Resp. | 28 | 32 | 28 | 28 | 40 | 36 | 52 | 28 |
| Bowels | 3 | 2 | 3 | 2 | 2 | 3 | 3 | 3 |
| Weight | 10 lbs. | | | | | | | |

Vomits. Saline Adrenalin Infusion 3vii 2 Saline Adrenalin Infusion 3vii 2 Saline Adrenalin Infusion 3vii 0 Saline Adrenalin Infusion 3vii 0 Saline Adrenalin Infusion 3vii 2 Saline Adrenalin Infusion 2 4

Hypo. Saline 5 c.c. bis die

| Date | Diet | Treatment |
|---|---|---|
| August 19 | Albumen Water 3ii 2 hrly | Stomach and Rectal lavage daily. |
| | Water at intervals. | Saline Adrenalin Infusion 3iv |
| 20 | | Rectal Injection Protargol 0.5% daily. |
| | | Strych. inj. hyp. 1 in 100 mj ij - hrly for 4 hrs |
| 21 | | Tinct. Opii - mj/4. by mouth B.P. |
| 10 pm. | | Saline Adrenalin Infusion 4 hrly. 3v |
| 22 | | Normal Horse Serum - 5 c.c. bis die. |
| | | Omit Tinct. Opii. |
| 23 | White of egg per rectum 6 hrly. | Strych. inj. hyp. mj 2 hrly. |
| | | Saline Adrenalin Infus. 3iv mjiij of 1 in 1000 Adrenalin. |
| 24 8 pm | Whey 3½ hrly cold. Omit. Alb. Water 3i hrly cold. | |
| 25 | White of egg per rectum 6 hrly. | 12.30 a.m. Tinct. Opii. B.P. mj/4 one dose. |

| Date | Diet | Treatment |
|---|---|---|
| August 26 | Whey 3½ hrly | |
| | Brandy mjx hrly. | |

feed. Physical examination proved negative. He rallied somewhat after saline adrenalin infusion but the temperature remained high. Under Horse Serum he seemed to improve temporarily, and on August 24th, he was decidedly better, but then the vomiting and diarrhoea became much more acute and he died two days later. A post mortem examination was not obtained.

In this case three observations were taken of the opsonic index before the horse serum treatment was begun. The average was low, between 0.30 and 0.52. For twenty four hours it remained low, then a temporary rise to 0.74 was seen but next day it had fallen to 0.48. The only remark I would make on this experiment is to note the general low tendency of the index. I cannot attribute with any certainty the one rise observed to the effect of the horse serum (Chart No. 34 ).

Case 23 , Ivor Everard, 4/12; diarrhoea and vomiting 3 weeks ago which lasted 14 days; vaccinated August 18th. Diarrhoea suddenly reappeared August 20th. Bottle fed from birth - cow's milk and barley water; at two months Patent Barley added

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 25 | Ivor Everard | 4/12 | Goldsmith | |

DISEASE

Result Death.

| Date August | 25 | 26 | 27 | 28 | 29 | 30 |
|---|---|---|---|---|---|---|
| Pulse | 130 | 136 | 132 | 120 | 138 | 150 |
| Resp. | 36 | 32 | 32 | 30 | 24 | 40 |
| Bowels | 1 | 3/4 | 2/3 | 3/2 | 3/2 | 3 |

Weight 9 lbs. 4

Vomits 3 3 1 3 1 2 6 1 6

Saline Adrenalin 3vi

Aug. 25 Normal Serum 5 c.c. mg. bis die

| Date | Diet | Treatment |
|---|---|---|
| Aug. 25 | Whey 3 i hrly. | Stomach and rectal lavage |
| 8 pm. | omit Whey. Albumen Water 3 i Cold. hrly | Serum 5 c.c. bis die. |
| Aug. 26 | Alb. Water 3 i hrly Cold. Nasal feeds. | |
| | | Aug. 27. Saline Adrenalin Infusion 3 vi |
| Aug 29 | Brandy m X Feed 2 hourly. White of two eggs peptonised per rectum | Strychnine Liq. hypo. m i 2 hrly. Tinct Iodi m i Glycerine m X Aquam ad 3 i hrly for 3 doses. |

| Date | Diet | Treatment |
|---|---|---|
| | | |

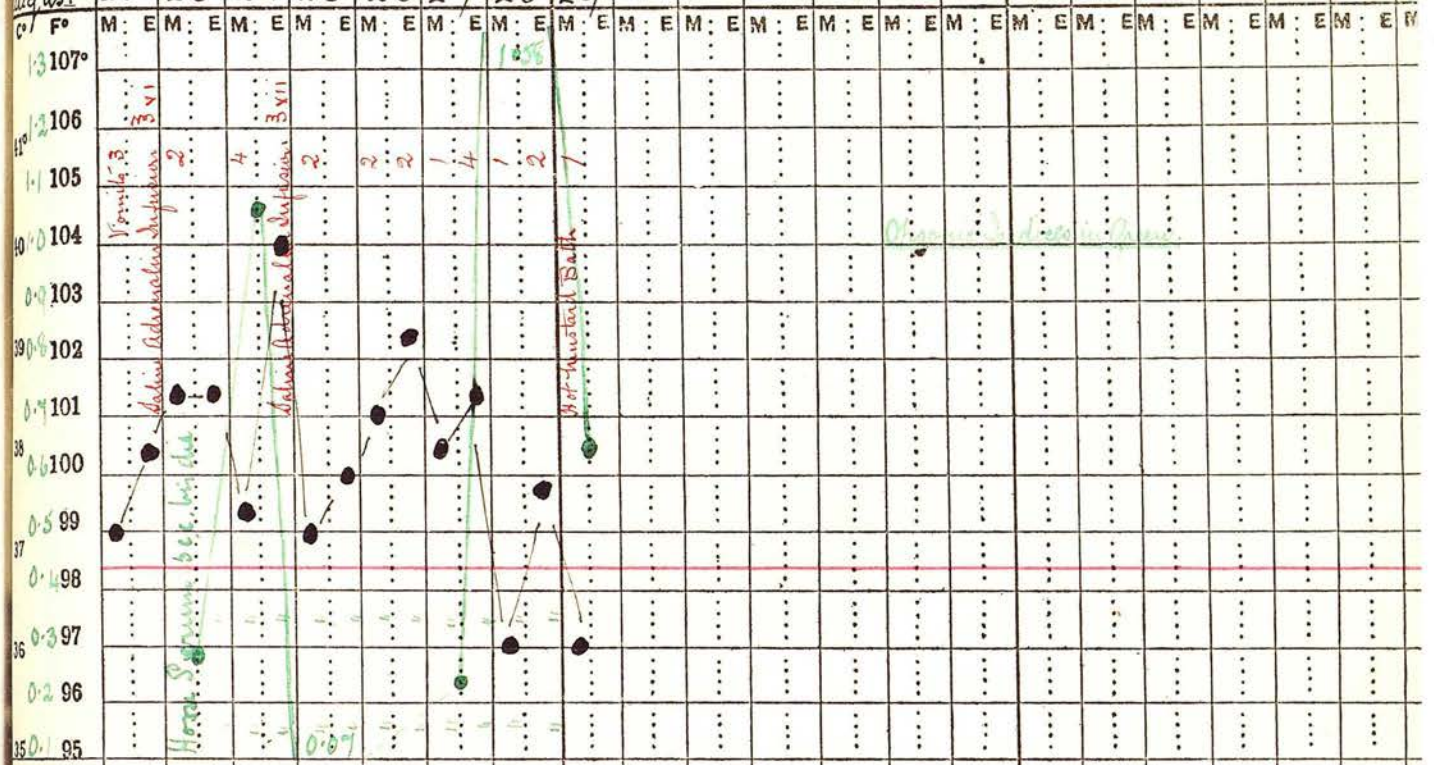
to diet.

Physical Examination negative ; buttocks not erythematous. Motions - frequent - green - fluid - consist of blood and slime; contain Morgan's Bacillus. Patient was admitted in a collapsed condition. After lavage he rallied slightly, but then vomiting became so troublesome as to necessitate nasal feeding. He died six days after admission and no light was thrown on the case by the post mortem examination.

Horse Serum was given from the first day and no normal was taken. The first observation was three days after admission, 0.28; that night a saline adrenalin infusion was given; next day the opsonic index had risen to 0.80 and the patient was somewhat better but this rise was not maintained. The only point of interest in this case is the probable causal connection between the improvement resulting from a saline adrenalin infusion and a rise in the opsonic index, (See Chart 25)

Case, 24 Florence Russell, 5/12; diarrhoea for 7 days; vomiting for 6. Breast fed for one month; since scalded cow's milk and barley water freshly

| No. 26 | Name Flo. Russell | Age 5/12 | Ward Goldsmith | Under Care of |
|---|---|---|---|---|
| Disease | | | | Result Death. |




| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Pulse | 128 | 120 | 140 | 128 | 140 | 160 | 128 | 156 |
| Resp. | 48 | 36 | 32 | 32 | 28 | 52 | 28 | 36 |
| Bowels | 1 | 4 / 3 | 4 / 2 | 3 / 4 | 2 / 4 | 3 / 4 | 3 / 3 | 4 |
| Weight | | | 8lbs. 4. | | | | | |

| Date | Diet | Treatment |
|---|---|---|
| August 22 | Alb. Water ℥ii 2 hrly. | Stomach and Rectal lavage daily |
| | | Saline Adrenalin Infusion ℥vi |
| 23 | Whey ℥ii 2 hrly | Horse Serum - 5 c.c. bi die. daily. |
| 10 pm. | Whey ℥i hrly cold. Brandy mx | Strych. inj. hyp. - 1 in 400 mij 2 hrly. for 6 doses. |
| 24 | White of egg per rectum 6 hrly. | 6 pm. Saline Adrenalin Infusion ℥vii |
| | | 11 pm. Rep. Strych |
| 25 | Omit egg. (5 given) Albumen water ℥i hrly cold. | |
| 27 | Alb. Water ℥i hrly iced. | |
| | midnight White of egg per rectum 6 hrly. | |
| 28 | | Tinct. Opii - B.P. m 1/4 : 4 hrly |

| Date | Diet | Treatment |
|---|---|---|
| Aug. 29 | | Hot mustard Bath. |
| | | Oxygen. |
| | | Omit Strych. |
| | | { Tinct. Iodi mx<br>Glycerini mv<br>Aquam ad ℥i |
| | | one dose. |

made for each feed. On admission the patient was so collapsed as to need infusion; the motions were dark green, foetid and frequent, but did not contain Morgan's bacillus; she was vomiting occasionally; the buttocks were erythematous but otherwise the physical examination proved negative. She rallied on the second day but the improvement was not maintained. The motions continued frequent, 6-8 per day, and green, and the vomiting was very troublesome. I would like to call attention to the fact that a minim of Tinctura Iodi was given just before death, for a very curious gastric condition was found post mortem, (See Chart 26).

Post mortem:

Ears:- Muco pus in both - membrane not congested nor perforated.

Abdomen:- On opening abdomen the transverse color was seen to be very distended and to bulge upon the other viscera; the descending was also distended and to a less extent the ascending.

Stomach:- Very contracted - walls very hard at pyloric end - flaccid and in folds at the cardiac end. In direction the viscus descended more or less vertically from the cardia, but midway it made a sudden swerve to the right passing horizontally

Stomach showing pyloric canal
of His in systole

Florence Russell
Case 24

to the pylorus showing seemingly the "pyloric canal of His"in a condition of "systole" Pyloric orifice was normal.

Intestine - Small - Empty and collapsed with thin, white walls.

Intestine, Large - Colon and sigmoid dilated - follicles enlarged; at the lower part there was a gelatinous condition of the lining membrane which peeled off easily leaving the muscular layer exposed.

Horse Serum was given from the second day, but no normal was taken. The Chart( No 26 ) shows a most remarkable series of readings. There were two well marked positive phases with a profound negative phase intervening. The first positive phase followed upon the initial dose of horse serum, but what was the reason of the subsequent immediate and profound fall? And what brought about the ante mortem rise? Was the latter due to the development of the inflammatory condition which after death was found in the ears?

Case 15 , Albert Quiver, 5/12; this case has already been detailed (Page 106 ). It was a case of pneumococcal meningitis occurring along with

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 27 | Eva Evans | 8/12 | Goldsmith | |

Disease

Result Death.

| Date | Aug 27 | 28 | 29 | 30 | 31 | Sept 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 130 | 120 | 88 | 100 | 136 | 144 | 124 | 112 | 136 | 144 | 148 | 136 |
| Resp. | 36 | 32 | 24 | 28 | 36 | 44 | 40 | 44 | 36 | 36 | 30 | 36 |
| Bowels | | 3/2 | 3/3 | 2/2 | 3/3 | 3/ | 2/1 | 2/4 | 2/4 | 2/3 | 2/4 | 3/4 |
| Weight | | | | | | 8 lbs. 2 | | | | | 8 lbs. | |

Vomit. 2. Saline Adrenalin Infusion 3 vii

0·07

Horse Serum see Kardex

Organic Lesions in Green

| Date | Diet | Treatment |
|---|---|---|
| Aug. 27 | Alb. Water 3ii<br>Brandy m XV 2 hrly. | Aug 27 Stomach and rectal lavage daily.<br>Saline Adrenalin Infusion 3 vii |
| " 30 | Lactated milk 3i<br>Boiled Water 3i 2 hrly | Sept: 2 Protargol 0.5% per rectum 3 iv |
| Sept: 3 | Lactated milk 3vi<br>Boiled Water 3iv 2 hrly. | Sept: 1. Normal Horse Serum 5 c.c. bisdie. |
| " 4 | Alb. Water 3ii<br>Fed nasally. 2 hrly | Sept: 3 omit Protargol per rectum.<br>Protargol grs 1/8 4 hrly. by mouth |
| " 6 | Lactated milk 3vi<br>Boiled Water 3i 2 hrly<br>Brandy m XX | Sept: 5 Strychnine - 1 in 400 — m iii m/p. for one dose.<br>Rep. in m ii 2 hrly for 6. |
| | | Sept: 6 Rep. Strychnine. |

diarrhoea. There was a well marked ante mortem rise to 1.61. Horse Serum was given throughout.

Case 25 Eva Evans, 8/12; diarrhoea and vomiting for 5 days. "Never strong"; diarrhoea and vomiting when 5 months old for one week. Breast fed for 3 weeks, then bottle - cow's milk and barley water. Weight 8 lbs. 8. On admission the patient was so collapsed as to necessitate a saline adrenalin infusion. The liver and spleen were enlarged; she had only 2 teeth; there was slight bronchitis at the right base; the temperature was markedly febrile and there was no ear condition present. The motions were frequent, about 6 per day; they were at first green and then colourless; and contained Morgan's bacillus. For details of treatment see Chart 27. The opsonic index remained remarkably low throughout. After obtaining a normal from three examinations August 28, 29 and 30, Horse Serum was first given on September 1. (Charts 27 & 28.) No appreciable difference is noticeable on the opsonic Chart. It remained uniformly low with but a slight tendency to an ante mortem rise.

| No. | Name | Age | Ward | Under Care of |
|---|---|---|---|---|
| 28 | Marie Cameron | 2/12 | | |

Disease

Result Death

| Date | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 24 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| August | | | | | | | | | | | | | | | | | | | | |
| M. | 3 | 0 | 0 | 0 | 2 | 1 | 5 | 2 | 0 | 0 | 0 | 0 | 3 | 1 | 0 | 0 | 2 | 1 | 0 | 2 |
| E. | | | | | | | | | | | | | | | | | | | | |

Temp. C° / F°: 41° 106 · 40 104 · 39 102 · 38 100 · 37 98 · 36 96 · 35 95

Vomits (4th)

Normal Saline Infusion 3iv (18th)

Organic Index

Pulse

Resp.

Bowels

| Weight | lbs. | lbs. oz. | lbs. oz. | lbs. oz. |
|---|---|---|---|---|
| | 7 | 7.10 | 7.6 | 6.8 |

| Date | Diet | Treatment | Date | Diet | Treatment |
|---|---|---|---|---|---|
| | | | | | |

Case 26. Marie Cameron, 2/12: vomiting and diarrhoea of 3 days duration. Breast fed for first 6 weeks, then Nestle's milk and prepared barley. For the past month she has been vomiting and wasting. On admission the child was very feeble; too weak to suck; had to be spoon fed. There were no adventitious sounds in the chest. Neither liver nor spleen were enlarged. Weight only 7 lbs. On routine treatment, her motions became yellow, the vomiting ceased and she gained 10 ozs. in weight. But on August 9th she began to relapse, the stools became green again and frequent. The temperature became febrile; she gradually sank and died on August 24th. From August 19th she was kept alive by infusions of normal saline.

Normal Horse Serum has been much lauded by many authors, notably Metchnikoff[1] for various conditions. Le Ray Reinach,[2] and others have used it extensively in this disease and claim to have had excellent results. I was very disappointed with my experience of it. According to Hoppe-Seyler 10 cubic centimetres of horse serum contain 1·5 grams of albumen which corresponds to 50 grams of cows milk and to 150 grams of maternal milk. Landois has shown that the injection of blood. serum increases proteid metabolism and raises the total amount of urea excreted. But although a priori it seems to be an ideal substance for temporising yet serveral of my cases died within twenty four hours, and those which I have here described did not seem materially influenced. All the cases died. I have discussed briefly the opsonic charts. The opsonic indices were taken to Bacillus Coli Communis. Our normal was the index of my blood and that of Dr. Taylor to whom I am indebted for much assistance and many of these observations. /

1. Annals de l'Institut Pasteur. ——— K
2. Journal de Medecine de Bordeaux 1905.

observations. Negative and positive phases occurred with apparent indifference to the exhibition of Horse Serum. In most of the cases the opsonic index gives us no indication of the patients impending death but in several it was remarkably and constantly low. The antemortem rise which occurred in five of the cases is not very easy to explain. The opsonic index afforded us no indications for treatment excepting the notable frequency with which a marked rise occurs after infusion either with normal saline alone or with that to which adrenalin has been added.

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